INDOOR AIR POLLUTION

IN MASSACHUSETTS

Final Report

The Commonwealth of Massachusetts
Special Legislative Commission
on Indoor Air Pollution

April 1989

COMMONWEALTH OF MASSACHUSETTS

SPECIAL COMMISSION ON INDOOR AIR POLLUTION

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Appointed by the President:
Senator William B. Golden, Senate Chair
Senator Carol C. Amick
Senator Lois G. Pines
Senator Peter C. Webber

Appointed by the Speaker:
Representative Patricia A. Walrath, House Chair
Representative Steven Angelo
Representative Carol C. Cleven
Representative Christopher J. Hodgkins
Representative John H. Loring
Representative Joseph B. McIntyre

Howard S. Wensley - Department of Public Health
(designee of the Commissioner of Public Health)

Sarah Simon - Department of Environmental Quality Engineering
(designee of the Commissioner of Environmental Quality Engineering)

Richard Levine - Department of Labor and Industries
(designee of the Commissioner of Labor and Industries)

David C. Macartney - State Board of Building Regulations
(designee of the Chairman of the State Board of Building Regulations)

Norman Anderson - Environmental Protection Agency
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Appointed by the Governor:

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(member of the American Lung Association of Massachusetts)

Barbara A. Kern - Massachusetts Association of Health Boards
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Phyllis M. Boucher - Massachusetts Health Officers Association
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P. Barry Ryan - Harvard School of Public Health
(academician from a local college or university with experience in the area of indoor air pollution and mitigating its effects)

Joseph L. Kociubes - Bingham, Dana and Gould
(representative of the building materials industry)

Donald F. Steele - AIRXCHANGE, Inc.
(representative of the heating and ventilating industry)

David W. Bearg - Life Energy Associates
(expertise in indoor air pollution mitigation)

INDOOR AIR POLLUTION

IN MASSACHUSETTS

FINAL REPORT OF

THE SPECIAL LEGISLATIVE COMMISSION

ON INDOOR AIR POLLUTION

Under Chapter 10 of the Resolves of 1986,

Chapter 2 of the Resolves of 1987

and Chapter 2 of the Resolves of 1988

ACKNOWLEDGMENTS

The Special Commission wishes to thank the following for their generous contribution of time and talent: Al Comproni and Robert Hallisey (Department of Public Health); Davida Andelman and Kathie Mazer (American Lung Association); Bede Wellford (AIRXCHANGE); Kathy Norman (Senator Golden's Office); and Joan O'Brien (Representative Walrath's Office).

The Commission wishes to pay special tribute to Sally Zielinski for writing the interim report; Elizabeth Conklin, Research Director of the Commission until August 1988; Colleen Ottoson and Norma Bourgeios for their assistance in preparing the final report.

Finally, the Commission would like to express its sincere gratitude to Donna Vorhees (McGregor, Shea & Doliner, P.C.) for her contribution and expertise in writing the final report.

LETTER OF TRANSMITTAL

To The Honorable Senate and The House of Representatives:

We, the undersigned, having voted in the affirmative, do hereby transmit this final report on the results of the investigation and study authorized under the provisions of Chapter 10 of the Resolves of 1986, Chapter 2 of the Resolves of 1987 and Chapter 2 of the Resolves of 1988.

Respectfully Submitted,

William B. Golden, Senate Chair

Patricia A. Walrath, House Chair

Lois G. Pines

Steven Angelo

Peter C. Webber

Carol C. Cleven

Howard S. Wensley

Christopher J. Hodgkins

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Chapter 10

THE COMMONWEALTH OF MASSACHUSETTS

In the Year One Thousand Nine Hundred and Eighty-six

RESOLVE PROVIDING FOR AN INVESTIGATION AND STUDY OF THE PUBLIC HEALTH EFFECTS OF INDOOR AIR POLLUTION.

RESOLVED, That a special commission consisting of four members of the house of representatives, three members of the senate, the commissioner of the department of public health or his designee, the commissioner of the department of environmental quality engineering or his designee, the commissioner of the department of labor and industries or his designee, the chairman of the state board of building regulations or his designee, the Regional Administrator (Region I) of the U.S. Environmental Protection Agency or his designee and seven persons to be appointed by the governor, one of whom shall be a member of the American Lung Association of Massachusetts, one of whom shall be a member of the Massachusetts Association of Health Boards, one of whom shall be a member of the Massachusetts Health Officers Association, one of whom shall be an academician from a local college or university with expertise in the area of indoor air pollution and mitigating its effects, one of whom shall be representative of the building materials industry, one of whom shall be a representative of the heating and ventilating industry, and one of whom has expertise in the area of indoor air pollution mitigation, is hereby established for the purpose of making an investigation and study of the public health effects of indoor air pollution, so called, including but not limited to, the effects of natural emissions of radon, the emissions from building materials such as urea formaldehyde and asbestos, the effects of emissions from wood stove, coal stoves, and fireplaces and the relationship between ambient air pollution and indoor air pollution. The commission shall also study the effects of personal habits, such as smoking and the relationship of indoor air pollution and ener-

gy conservation measures. Said commission shall report to the general court the results of its investigation and study, and its recommendations, if any, together with drafts of legislation necessary to carry such recommendations into effect by filing the same with the clerk of the house of representatives on or before the first Wednesday in November, nineteen hundred and eighty-seven.

House of Representatives, December 11, 1986.

Passed, George Keverian, Speaker.

In Senate, December 11, 1986.

Passed, William M. Bulger, President.

December 23, 1986.

Approved,

Governor.

LIST OF ACRONYMS AND TECHNICAL TERMS

ach	air changes per hour
ACGIH	American Conference of Governmental Industrial Hygiene
ACM	asbestos-containing material
AHERA	Asbestos Hazard Emergency Reponse Act
AIHA	American Industrial Hygiene Association
ASHAA	Asbestos School Hazard Abatement Act
ASHRAE	American Society of Heating, Refrigeration and Air Conditioning Engineers
B(a)P	Benzo-a-pyrene
BOH	board of health
BPA	Bonneville Power Administration
BPD	bronchopulmonary dysplasia
CERCLA	Comprehensive Environmental Response, Compensation and Liability Act (Superfund)
cfm	cubic feet per minute
CFR	Code of Federal Regulations
CIAQ	Committee on Indoor Air Quality
CIIT	Chemical Industry Institute of Toxicology
CMR	Code of Massachusetts Regulations
CO	carbon monoxide
CO2	carbon dioxide
CPSC	Consumer Product Safety Commission
DAQC	Division of Air Quality Control
DCPO	Massachusetts Division of Capital Planning and Operations
DEQE	Massachusetts Department of Environmental Quality Engineering
DFA	Massachusetts Department of Food and Agriculture
DHHS	Department of Health and Human Services
DLI	Massachusetts Department of Labor and Industries
DNA	deoxyribonucleic acid
DOE	Department of Energy
DOH	Massachusetts Division of Occupational Hygiene
DOT	Department of Transportation
DPH	Massachusetts Department of Public Health
EPA	Environmental Protection Agency
ETS	environmental tobacco smoke
f/cc	fibers per cubic centimeter
FDA	Food and Drug Administration
FEMA	Federal Emergency Management Agency
FEV	forced expiratory volume
FHA	Federal Housing Administration
FIFRA	Federal Insecticide, Fungicide and Rodenticide Act
FR	Federal Register
FTC	Federal Trade Commission
FWPCA	Federal Water Pollution Control Act
HCl	hydrogen chloride
HUD	Department of Housing and Urban Development
HVAC	heating, ventilating and air conditioning
IPM	integrated pest management

IRIS	Integrated Risk Information System
LEA	local education authority
MDF	medium density fiberboard
MS	mainstream smoke
NAAQS	national ambient air quality standards
NAS	National Academy of Sciences
NCI	National Cancer Institute
NCRP	National Council on Radiation Protection and Measurement
NESCAUM	Northeast States for Coordinated Air Use Management
NESHAPS	National Emission Standards for Hazardous Air Pollutants
NIH	National Institute of Health
NIOSH	National Institute for Occupational Safety and Health
NOx	nitrogen oxides
NO2	nitrogen dioxide
NRC	National Research Council
NUPAS	National Urban Pesticide Applicators Survey
ORNL	Oak Ridge National Laboratory
OHER	Office of Health and Environmental Research
OSHA	Occupational Safety and Health Administration
PAH	polycyclic aromatic hydrocarbons
PCM	phase contrast microscopy
pCi/l	pico Curies per liter
PEL	permissible exposure level
PF	phenol formaldehyde
PLM	polarized light microscopy
ppb	parts per billion
ppm	parts per million
RCP	Massachusetts Department of Public Health, Radiation Control Project
RCRA	Resource Conservation and Recovery Act
RH	relative humidity
RMP	Radon/Radon Progeny Measurement Proficiency
RSP	respirable particulates
SARA	Superfund Amendment and Reauthorization Act
SBS	sick building syndrome
SEM	Scanning Electron Microscopy
SDWA	Safe Drinking Water Act
SIDS	sudden infant death syndrome
SO2	sulfur dioxide
SS	sidestream smoke
TEM	transmission electron microscopy
TSCA	Toxic Substances Control Act
UF	urea formaldehyde
UFFI	urea formaldehyde foam insulation
ug	microgram
UMTRCA	Uranium Mill Tailings Radiation Control Act
VOC	volatile organic compound
WL	working level
WLM	working level month

RESOLVE INCREASING THE MEMBERSHIP OF THE COMMISSION

H 4812

Chapter 2.

THE COMMONWEALTH OF MASSACHUSETTS

In the Year One Thousand Nine Hundred and Eighty-seven

RESOLVE INCREASING THE MEMBERSHIP OF THE SPECIAL COMMISSION ESTABLISHED TO MAKE AN INVESTIGATION AND STUDY OF THE PUBLIC HEALTH EFFECTS OF INDOOR AIR POLLUTION.

RESOLVED, That the membership of the special commission, established by chapter ten of the resolves of nineteen hundred and eighty-six, is hereby increased by two members of the house of representatives and one member of the senate.

House of Representatives, July 9, 1987.

Passed, George Keverian, Speaker.

In Senate, July 9, 1987.

Passed, Walter J. Boveri, Acting President.

July 21, 1987.

Approved,

Michael S. Dukakis, Governor.

RESOLVE CONTINUING THE COMMISSION

H 5455

Chapter 2.

THE COMMONWEALTH OF MASSACHUSETTS

In the Year One Thousand Nine Hundred and Eighty-eight

RESOLVE REVIVING AND CONTINUING THE SPECIAL COMMISSION ESTABLISHED TO MAKE AN INVESTIGATION AND STUDY OF THE PUBLIC EFFECTS OF INDOOR AIR POLLUTION.

RESOLVED, That the special commission, established by chapter ten of the resolves of nineteen hundred and eighty-six, is hereby revived and continued.

House of Representatives, July 16, 1988.

Passed, Acting, Speaker.

In Senate, July 16, 1988.

Passed, William M. Bulger, President.

July 26, 1988.

Approved,

Governor.

EXECUTIVE SUMMARY

INDOOR AIR POLLUTION

The indoor air we breathe often contains pollutants which may have health effects ranging from annoying to deadly. Major pollutant types found in indoor environments include tobacco smoke, radon gas, formaldehyde, asbestos, volatile organic compounds, pesticides, combustion products and biological contaminants. For most of these pollutants, concentrations measured indoors exceed levels found outdoors yet current environmental air pollution laws and regulations are not protective of these indoor environments. They focus instead on the outdoor environment even though individuals spend about ninety percent (90%) of their time indoors.

The Special Legislative Commission on Indoor Air Pollution was established in 1986 to respond to the indoor air pollution threat. The Commission's mandate has been to investigate and study public health effects of indoor air pollution and to report their findings and recommendations to the General Court. The Commission has conducted its own research effort to determine the health effects associated with indoor air pollutants and has also drawn upon the expertise of numerous New England consultants and professors knowledgeable about specific aspects of the problem.

The Commission's efforts confirm the seriousness of the indoor air pollution health threat, which worsened with the energy conservation efforts of the 1970s. More insulation and tighter construction led to lower ventilation rates and build-up of contaminants. Many "sick" buildings have been identified where occupants suffer severe or recurring discomforts such as headaches, dizziness, fatigue, eye irritation, and respiratory problems. Other conditions attributable to indoor air contaminants include: cancer; bronchitis; pneumonia; heart, circulatory and respiratory problems; impaired vision; skin rash; chemical sensitivity; birth defects; and mental, nervous and immunological disorders.

Indoor air quality measurement and monitoring is often difficult, with few standard methods available. These data are needed to develop risk profiles for various indoor pollutants. Risk profiles are based upon the hazardous nature of the substance, how a health effect varies with the concentration or total exposure to the substance and whether concentrations or total exposures of concern occur in indoor air. Risk profiles have been attempted for several pollutants, but for the most part, little is understood about the magnitude of indoor air pollution health risks. Nevertheless, substantial evidence exists which confirms that these risks are serious enough to warrant some form of action. Available data and information provide adequate guidance for the Commission to set priorities for addressing the indoor air pollution problem.

The Commission has found that numerous indoor air quality pollution mitigation options are available. Elimination of the source provides the most effective means of mitigation but is often impractical. Controlling the source through emission limitations or by educating the occupants about its

safe use may further reduce indoor air pollution. Removal or dilution of pollutants in indoor air using ventilation and air cleaning systems is also effective and need not conflict with the goal of energy conservation given the availability of ventilation systems that recapture heat from vented air.

Policy decisions surrounding regulation of indoor air are complex but key to determining government's role in improving indoor air quality. In Massachusetts, several agencies have authority over specific indoor air quality problems, while local health authorities play an important role in protecting the public health and safety generally from indoor air health threats. Within the current federal, state and local regulatory structure, individual programs and authorities must be strengthened and their efforts coordinated to ensure comprehensive and effective control over indoor air pollution.

GENERAL COMMISSION RECOMMENDATIONS

The Commission views indoor air pollution as a serious public health problem necessitating government regulatory action. Recommendations of the Commission are designed to maximize protection of the public health through control of indoor air pollution, while minimizing intrusion into decision-making about privately-owned buildings. The Commission identified few indoor air jurisdictional gaps, but rather many areas where programs and authorities could be strengthened.

So that state regulation of indoor air quality is comprehensive and well organized, the Commission supports the establishment of an Interagency Coordinating Council to address indoor air health issues by defining appropriate state actions for prevention and remediation of health threats.

The Commission also recognizes the need for and fully supports public education efforts concerning indoor air pollutants, their sources and how these sources can be controlled. The goal of such a program is not only to increase awareness of the indoor air pollution problem, but to equip individuals with the knowledge they need to choose household products and building materials and to design and maintain buildings so as to minimize health risks.

The Commission has found that where source control is impractical, properly designed and maintained ventilation systems represent the predominant means for controlling indoor air pollutant levels. Ventilation systems can effectively control all types of indoor air pollution. The Commission therefore supports legislation that establishes state-of-the-art standards for ventilation systems in public buildings and in new or substantially renovated private buildings. These standards address both system design and long term maintenance. Commission recommendations specific to various indoor air pollutants are included in the following sections.

RADON

Radon gas emitted from uranium-bearing rock and soil is associated with increased incidences of lung cancer in miners. Concern about health risks of radon gas arose when elevated levels of radon were found inside homes built on mine wastes and later in homes on undisturbed land. Levels of indoor radon are a function of several factors including the amount of uranium in the local substrate, soil permeability, the pathways by which radon enters the stuctures and the living patterns of the residents. Radon can enter buildings as a gas from the soil via gaps in the structure, from tap water which has passed through uranium-bearing rock, or in uranium-containing construction materials such as stone in a fireplace. Indoor levels of radon may also be elevated due to "radon-daughters," radioactive radium decay products, attaching to ambient particulates of environmental tobacco smoke. It appears that elevated radon levels can be mitigated through modifications in structures, ventilation and living patterns.

Indoor radon levels at concentrations above the Environmental Protection Agency's (EPA) action level of 4 pico Curies per liter (pCi/1) annual average exposure have been found in many states, including Massachusetts. The bedrock and surficial geology of the Commonwealth indicate the potential for moderate to high radon levels in some areas. In others the rock type is usually low in uranium content but may have local hot spots. Through the efforts of this Commission, funding was obtained for a radon study jointly conducted by EPA and the Department of Public Health (DPH). This study found that nearly one fourth (1/4) of all homes tested in Massachusetts had screening levels of radon in excess of EPA's action level.

The risk of dying of lung cancer from long term low level radon exposure is based on extrapolation from the effects of the high doses received by miners. A few structures have levels comparable to mines. Current data indicate a 1% to 5% probability of developing lung cancer from a seventy (70) year exposure to 4pCi/L concentration of radon.

Lack of a comprehensive program at either the state or local level makes action to address indoor radon a matter of some urgency. The Commission therefore recommends the following:

- Passage of proposed legislation which would require disclosure of any radon test results performed on a building, condominium or apartment unit at the point of sale;

- Increase in public awareness of the radon problem;

- Change the State Building Code to avoid radon intrusion into newly constructed or substantially renovated buildings and to increase the required ventilation rate; and

- Develop a certification program for contractors performing radon mitigation.

FORMALDEHYDE

Formaldeyde is a reactive gas with a pungent odor which has been associated with health effects ranging from irritation of the eyes, skin and respiratory system, headache and nausea to potential cancer. Formaldehyde is believed to cause nasal cancer in animals and there is limited evidence of its carcinogenic effect in humans. EPA has designated formaldehyde a probable human carcinogen and the Department of Environmental Quality Engineering (DEQE) has researched the need for outdoor air quality limits believed to be protective of human health.

Formaldehyde is ubiquitous in the indoor environment. Major sources in indoor air are cigarette smoke and bonded wood products made with urea formaldehyde resins. Plywood and particleboard are in wide use in furniture, cabinets, flooring, urea formaldehyde foam insulation (UFFI) and wall paneling. Formaldehyde is also used to treat textiles used for clothing, draperies and upholstery for crease resistance, colorfastness, shrink resistance, water- and flame-proofing. Formaldehyde is a product of hydrocarbon combustion and therefore occurs in indoor air where gas stoves and heaters are improperly vented or where people smoke tobacco products.

Elevated levels of formaldehyde may occur in mobile homes that contain large quantities of bonded wood products and are poorly ventilated, in tightly constructed new homes or in biological laboratories and industrial facilities where formaldehyde is used. Formaldehyde concentration in a room is determined by the nature of the emitting material, its age, temperature, humidity, and ventilation.

The Occupational Safety and Health Administration (OSHA) has reduced its workplace standard for an 8-hour average exposure to formaldehyde from 3 to 1 ppm. Both the American Society of Heating, Refrigeration and Air Conditioning Engineers (ASHRAE) and the National Institute for Occupational Safety and Health (NIOSH) have adopted 0.1 ppm as a comfort-based indoor air quality guideline for formaldehyde.

The Housing and Urban Development Agency (HUD) has developed product emission standards for bonded wood products in manufactured homes which are based upon assumptions regarding home design and ventilation. Spraying UFFI on the walls of over 400,000 homes nationally during the 1970s led to many complaints. Massachusetts banned UFFI in 1979. While a federal Consumer Product Safety Commission (CPSC) ban was overturned in court, UFFI is seldom used today. Massachusetts Legislation passed in 1985 established formaldehyde disclosure, testing and removal programs funded by industry contributions.

Commission recommendations for state action relative to formaldehyde include:

- Educating the public about formaldehyde sources and adverse human health effects;

- Supporting the reduction of the allowable formaldehyde indoor exposure level to below .1 ppm; and

- Providing for state-of-the-art ventilation system design and maintenance so as to minimize indoor formaldehyde levels.

ASBESTOS

Once considered a miracle fiber, asbestos is now widely recognized as a serious health hazard necessitating the expenditure of billions of dollars in remediation costs. Uses of asbestos fibers in the past ranged from the insulation of boiler and piping systems in ships to inclusion in building components as a fire retardant and an insulator from heat, cold, and electricity. Today, the use of asbestos is limited and highly restricted.

Certain health hazards attributable to exposure to airborne asbestos fibers include asbestosis, lung cancer and mesothelioma. Cases of asbestosis are most prevalent in industry workers exposed to asbestos and are characterized by pulmonary fibrosis, or the scarification of lung tissue. Asbestos fibers alone may cause lung cancer or they may act synergistically with cigarette smoking, multiplying the chances of developing lung cancer. Mesothelioma is a rare, almost always fatal cancer of the pleural or peritoneal cavities which is considered a marker for asbestos exposure.

Federal OSHA standards regulate asbestos levels in industrial settings while EPA has established specific standards under the Clean Air Act for demolition and renovation projects with procedures for asbestos emission control. EPA's recommended measure for proper levels during asbestos removal stands at 0.01 fibers per cubic centimeter for fibers greater than five microns in length. The Asbestos Hazard Emergency Response Act of 1986 (AHERA) requires the inspection of schools for the presence of asbestos and the preparation of asbestos management plans if asbestos is detected. Massachusetts has established a grant program to reimburse municipalities and school districts for 60-75% of the cost to remove, encapsulate or enclose asbestos found in private and public schools.

The Commonwealth's asbestos regulatory authority is distributed among three state agencies: the Department of Environmental Quality Engineering (DEQE), the Department of Labor and Industries (DLI), and the Department of Public Health (DPH). DEQE's asbestos regulations require notification to DEQE and strict work practices during building construction, renovation and demolition when asbestos is present. DLI's regulations also include these requirements as well as requirements recommended by the 1985 Asbestos Interagency Task Force for the licensing and certification of asbestos abatement contractors, workers and analytical laboratories. The DPH ensures that asbestos pipe, boiler and furnace coverings and insulation in residential settings are non-friable and that proper repair and removal of asbestos-containing materials are observed. State funds are available for asbestos abatement projects in certain schools and public buildings.

A recent EPA-sponsored study identified friable asbestos in 733,000 of 3.6 million public and commercial buildings in the U.S. with asbestos abatement costs estimated at 51 billion dollars. Given these results, EPA recommends acceleration of accreditation programs for asbestos abatement workers, better enforcement of existing asbestos-related rules and assessment of the continuing effort to abate asbestos hazards in schools.

To augment existing asbestos initiatives on the federal and state levels, the Commission proposes:

- Increase in public education regarding asbestos health hazards and abatement procedures;

- Establishment of asbestos-related training programs for local board of health officers and building inspectors;

- Improvement of staffing and resources within applicable state agencies; and

- Exploration of financing alternatives to bonding, such as letters of credit, for asbestos abatement contractors.

ENVIRONMENTAL TOBACCO SMOKE

Environmental tobacco smoke (ETS) is a pollutant most noticeable in confined, poorly ventilated indoor environments. Nonsmokers exposed to cigarette smoke face health effects similar to those commonly associated with cigarette smoking. While not identical, environmental tobacco smoke (ETS) shares many common health threatening substances with the cigarette smoke inhaled by smokers, although nonsmokers experience much lower doses.

Health effects include eye, nose and throat irritation, increased incidence of respiratory illness and decreased lung function in children, aggravation of asthma symptoms and lung cancer. A risk assessment conducted in 1985 estimated that aggregate exposure to ETS produces about 5000 lung cancer deaths per year in U.S. nonsmokers aged 35 years or older, with an average loss of life expectancy of 17 $\pm$ 9 years per fatality.

A 1986 U.S. Surgeon General report concluded the following: (1) Involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers; (2) Children of parents who smoke have an increased frequency of respiratory infections, increased respiratory symptoms, and slightly smaller rates of increase in lung function as the lung matures; and (3) Simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate, the exposure of nonsmokers to environmental smoke.

Federal regulation of ETS is sparse, with most regulatory activity occurring on the state level. Corporations are increasingly developing smoking policies. Some of these regulations and policies include total

smoking prohibitions in specific buildings, smoking prohibitions in certain areas of buildings or, at a minimum, requirements for the establishment of smoking policies agreeable to smokers and nonsmokers.

Massachusetts recently passed legislation banning smoking in many public places including elevators, retail food stores, school buses, supermarkets, courtrooms, public transit vehicles and other public areas. The Massachusetts DPH has developed a "Plan for Nonsmoking and Health," the primary goal of which calls for the reduction of tobacco use and increase of ETS control. DPH's goal for the year 2000 calls for more than 90% of adults and adolescents to be nonusers of tobacco and for nonsmokers to be able in the course of their normal daily activities to breathe ETS-free air.

Economic reasons for implementing smoking restrictions have been advanced in addition to health concerns. A DPH study estimates that in 1985 alone, smoking-attributable costs for Massachusetts totaled 1,597 billion dollars. The savings to employers has been estimated at 4,500 dollars per year for every employee that quits smoking, due to the reduction in absentee days, morbidity and premature mortality, insurance costs, lost time on smoking rituals, and property damage and depreciation.

In light of the public health and economic concerns surrounding ETS, the Commission recommends several statewide actions in addition to past legislative and agency efforts. Some of the more prominent recommendations include:

- Educating the public about the dangers of smoking in order to prevent nonsmokers from beginning to smoke;

- Limiting minors' access to cigarettes;

- Providing for state-of-the-art ventilation system design and maintenance so as to minimize indoor ETS levels.

- Implementing restrictions on smoking in hospitals, workplaces and retail outlets;

- Certifying new buildings or buildings that have been substantially renovated, for the purpose of determining whether the building standards have been met and whether retrofitting of ventilation systems should be required to protect occupants from ETS exposure; and

- Supporting the DPH's goal for the year 2000, calling for nonsmokers to be able in the course of their normal daily activities to breathe ETS-free air.

BIOLOGICALS

Biological contamination of indoor environments ranks third in NIOSH's list of indoor air health threats after poor ventilation and building fabric

contaminants. Types of biological contamination range from animal dander, pollen and dust to bacteria, fungi, and viruses. Poor ventilation, stagnant water in ventilation systems, and relative humidity levels falling outside the 40-60% range have been implicated as causes of high indoor air concentrations of these biological contaminants. High humidity and moisture concentrations support proliferation of biologicals in ventilation systems.

Some biological pollutants including house dust, pollen, animal dander and fungi, cause allergic responses and may even induce asthma. Certain viruses and bacteria cause infections and diseases such as Legionnaire's disease, Pontiac Fever and humidifier lung. Exposure to viruses and bacteria results from person to person contact, person to object contact, or inhalation of airborne organisms.

Biological contamination of indoor environments may be controlled by a number of means, including the regulation of relative humidity levels, proper design and maintenance of ventilation systems, overall cleanliness and periodic cleaning of all places where water is likely to collect.

Outside of the state DPH and local board of health general authority to protect the public health, very little regulation of indoor biological contamination exists. Given the severity of the problem and the lack of statewide attention to limiting indoor biological contaminant levels, the Commission has made several recommendations for action including:

- Development of a statewide tracking mechanism of efforts underway to study or minimize biological contamination;

- Establishment of better monitoring techniques, especially those with analytical methods that generate quick results; and

- Provision for state-of-the-art ventilation design and maintenance so as to minimize indoor biological contaminant levels.

COMBUSTION PRODUCTS

Combustion products emanating from gas and kerosene appliances, wood stoves, fireplaces, cigarettes and cars in attached garages pollute many indoor environments. Commonly detected combustion products include carbon monoxide, carbon dioxide, nitrogen oxides (NO_x), respirable particulates, sulfur dioxide, and polycyclic aromatic hydrocarbons (PAHs). The concentration of these combustion products largely depends upon the nature of the fuel used, the location of the combustion source, the quantity of air used, the temperature of combustion and the presence or absence of a ventilation system. Some particulates and PAHs have carcinogenic effects, while other combustion products such as carbon dioxide and carbon monoxide result in headaches and dizziness. Sufficiently high doses of carbon monoxide result in death. NO_x produces edema of the lungs, eye irritation and may increase airway resistance in humans. The EPA has established a NAAQS of 0.05 ppm as an annual average

for nitrogen dioxide (NO_2) based on such health effects. DEQE has established an hourly ambient guideline for NO_x of 170 ppb.

Woodsmoke poses not only an indoor health threat but an ambient environmental threat, thus federal performance standards have been set for particulate emissions from new woodstoves. Indoor levels of smoke are not regulated, yet woodsmoke leaks into indoor environments through improper woodstove installation, cracks in stovepiping, downdrafts and windows.

To minimize combustion product levels in indoor environments the Commission recommends the following:

- Concerted public education effort as to how combustion product levels may be eliminated or minimized through careful choice of appliances, design of woodstoves and proper ventilation system design and maintenance; and

- Revision of the state building code to ensure appropriate ventilation system design and maintenance to minimize indoor combustion product levels.

VOLATILE ORGANIC COMPOUNDS

Volatile organic compounds (VOCs) are the large category of carbon-based compounds that evaporate. They are ubiquitous in the modern indoor environment, found in particleboard, plastic and rubber solvents, household cleaners, air fresheners, paints and varnishes, furnishings and office equipment. Formaldehyde represents the most well-researched VOC in the indoor environment.

Health impacts associated with exposure to various VOCs range from eye and respiratory irritation to narcotic and carcinogenic effects. A recent study conducted by the EPA, the so-called "TEAM" Study, revealed the existence of much higher levels of VOCs indoors versus the outdoor environment, thus emphasizing the need to control indoor sources of VOCs. The Federal Interagency Integrated Chlorinated Solvents Project is comprised of various federal agencies regulating VOCs. The Project considers risks posed by certain VOCs in indoor environments with the goal of determining appropriate control and regulatory options for these substances.

The DEQE is investigating potential controls for VOC source emissions due to their contribution to unhealthful ozone levels. The DEQE is also participating in a regional effort to study how VOC emission from consumer products can be controlled and to investigate health effects associated with specific types of VOCs.

To minimize indoor VOC levels, the Commission recommends the following:

- Careful selection and use of household products, building materials and furnishings;

Maintenance of appropriate ventilation systems; and

Statewide public education compaign that describes VOCs, their health effects and how these effects may be mitigated.

PESTICIDES

Indoor levels of pesticide contamination originate from outdoor agricultural applications and lawn care as well as indoor plant care and pest control. They may rid the indoor environment of unwanted insects, fungi, or rodents but may also contaminate indoor air if not applied so as to eliminate or minimize human exposure.

By definition, pesticides are toxic. They may be absorbed through the skin, ingested through the mouth or inhaled into the lungs. Many are toxic to the liver, the nervous, respiratory and reproductive systems. Pesiticdes may also be carcinogenic, such as dichlorvos which is found in flea collars and no-pest strips.

The federal and state registration processes for pesticides are designed to prevent from entering the market those pesticides that pose an unreasonable adverse effect. Federal and state officials draw from information on the pesticide's chemistry, residue chemistry, wildlife and aquatic organism toxicology and specific risk data to make this determination. Many pesticides were registered prior to these federal requirements for toxicological and field testing, so EPA is now in the process of re-registering approximately 1400 pesticide active ingredients under a 1988 federal law. This process has been severely delayed, while pesticides posing significant public health threats remain on the market.

Two bills are pending in the Massachusetts legislature relative to pesticides. Both promote the use of Integrated Pest Management (IPM). IPM is generally defined as the means for controlling pests, the goal of which is to minimize reliance on chemical pesticides by using alternative natural methods.

Commission recommendations for pesticides include:

- Use of IPM and any other safe alternatives to chemical pesticide use;
- Education of the public relative to health threats posed by pesticides and the benefits of practicing IPM;
- Provision of public notice where pesticides are being applied; and
- Adoption of Department of Food and Agriculture guidelines for applying pesticides in occupied rooms as state regulations.

Chapter 1: INTRODUCTION

OVERVIEW OF PROBLEM

Indoor air quality is an emerging issue in Massachusetts, throughout the United States and in other industrialized countries. Reports of high radon levels in homes, nonsmokers suffering health effects from inhaling other people's tobacco smoke, sick buildings, and Legionnaire's disease mysteriously killing guests at a convention hotel have drawn attention to the problem of indoor air pollution.

Over the past several decades new substances have been identified in indoor air. Others have been found in unexpected concentrations. Higher levels of the same contaminant are often present indoors than outside, indicating that structures are no protection against air pollution. Most of these airborne compounds are colorless and odorless - undetectable to the senses.

Health effects associated with substances found in indoor air range from discomfort, such as nausea, headaches, eye and skin irritation, to chronic respiratory problems, cancer and mortality. For example, radon, formaldehyde and some components of tobacco smoke are suspected or proven carcinogens, and carbon monoxide from poorly vented stoves or heaters can cause brain dysfunction at low doses and death at high doses.

Since 90% of the average person's time is spent indoors, air quality in buildings is a public health issue which concerns not only owners and occupants but also government which must develop approaches to improve indoor air quality.

GOALS

The overall goal of the Special Commission on Indoor Air Pollution has been to develop a comprehensive approach to indoor air quality problems for the Commonwealth. Neither the federal government nor any state except California has a clearly structured, well coordinated program.

In response to the apparent magnitude and pervasiveness of indoor air pollution, the Commission's intent has been to provide the legislature with the information and, if needed, legislation to protect the public from the adverse health effects of air pollution by:
(1) establishing guidelines to achieve and maintain indoor air quality which protect occupants from the health effects of indoor air pollutants;
(2) designating a comprehensive program for indoor air quality;
(3) increasing public awareness of the indoor air pollution threat and how it may be eliminated or minimized.

ACTION PLAN

The Commission approached this complex topic by investigating indoor air quality as a whole, assessing individual categories of pollutants and the interactive effects of multiple contaminants, and identifying policy and regulatory issues and their resolution.

The many hazardous airborne gases, vapors and particulates found in indoor air have been grouped for convenience into the following categories: radon, formaldehyde and other volatile organic compounds (VOCs), asbestos, environmental tobacco smoke, pesticides, biological contaminants and combustion products. Investigations of each pollutant include characteristics and sources of the pollutant, health effects related to or suspected from exposure to the pollutant, history and scope of the problem, techniques of measurement and mitigation, risk to the population where such risk has been quantified, and existing regulatory approaches at the federal and state levels.

Data and information have been assembled from expert testimony, the scientific literature, certain secondary sources, Commission members and other interested participants.

INDOOR AIR POLLUTANTS: TYPES, SOURCES AND HEALTH EFFECTS

Health Effects

Indoor air pollution is a growing problem in the United States and accounts for up to 50% of all illnesses. Health costs are estimated at 100 billion dollars per year (1). Hazardous substances identified in indoor air include: radon, formaldehyde and other VOCs, asbestos, tobacco smoke, pesticides, biological contaminants, and combustion products.

The health effects of hazardous substances found in indoor air range from the irritating to the deadly depending on the substance, its concentration and the length of exposure. Effects may be immediate or delayed, and have acute or chronic health effects. Effects may vary both among individuals, based on sensitivity, and in the same individual at different times.

Long-term health effects of indoor air pollutants include cancer, birth defects, immunological problems, nerve damage, reproductive difficulties and developmental problems. Short term effects include pneumonia, upper respiratory infections, and allergic asthma (2, 3).

Even brief exposures to elevated concentrations of some pollutants, such as formaldehyde, carbon monoxide or nitrogen oxides, can lead to headaches, dizziness, nausea, eye irritation, and respiratory problems such as wheezing, coughing, congestion or shortness of breath. These symptoms often disappear when the source is removed (4, 5).

Assessment of the health impacts of indoor air pollutants may be complicated because the effects of some contaminants differ based on concentrations and duration of exposure. For example, a substance may have an offensive odor at low concentrations, but produce eye irritation at higher concentrations (6).

Some people are hypersensitive to a range of chemicals found in homes. They react, for example, to formaldehyde, petroleum products, cigarette smoke, and chemicals in building products like preservatives, dyes, plasticizers, and adhesives. Reactions range from mild discomfort to life threatening allergic reactions (7).

Most of the long range effects of airborne substances are known from workers who, following exposure to high levels on the job, developed a range of medical problems including respiratory diseases and cancer. Levels to which they were exposed were usually many times higher than levels to which people are exposed in non-occupational settings.

Little is known about long term, low level exposure as may occur in homes or workplaces (5). Until there is evidence to the contrary it is prudent to

assume that if a substance causes problems at high levels there could be a risk at low levels, especially if exposure occurs over a long period of time.

Categories of Pollutants

Radon

Radon is a radioactive gas arising from the natural decay of uranium in rock and soil. Radon gas may enter structures through pores and cracks or dissolved in well water. When radon-laden water emerges from a tap, radon gas escapes into the room air. Building materials made from rock containing uranium are an additional source. Structural elements such as stone foundations, walls or fireplaces may also produce radon (8). Levels above the EPA's recommended safe level for radon have been reported in many residences in the United States (9).

Inhalation of the radioactive compounds produced as radon spontaneously decays has been associated with lung cancer in miners (5). Risk estimates of the effects of long term low level exposure are widely accepted and of concern. Studies are underway to determine: areas of high radon potential, the factors contributing to high indoor radon levels, and the extent of elevated radon levels in the United States as well as the effects of long term low level exposures.

Formaldehyde

Formaldehyde is a pungent smelling gas which causes irritation of the eyes, skin and respiratory system, and is regarded as a probable carcinogen by the EPA. It has produced nasal cancer in animals (5). Major sources of formaldehyde in indoor air are bonded wood products and urea formaldehyde foam insulation (UFFI). Formaldehyde is also found in home furnishings such as carpets and draperies, and many other domestic products.

Bonded wood products, such as plywood and particleboard, are widely used as paneling, flooring, and in furniture and cabinets. Mobile homes have higher amounts of bonded wood products and higher formaldehyde levels than conventional homes. Homes may be grouped into three categories, based on method of construction: "conventional" homes which are built almost entirely on site; "mobile" homes which are provided with a chasis and transported to a site as an entity or in 2 or 3 parts; and "other manufactured" homes which include modular, paneled and kit built homes.

A recent EPA study showed that VOC levels indoors often exceed outdoor levels, largely due to building materials, household furnishings and cleaners (10). Elevated formaldehyde levels produced by emissions from UFFI led to a ban by the Consumer Product Safety Commission (CPSC) which was subsequently overturned in court. UFFI has been banned in Massachusetts since 1979.

Chapter 2: INDOOR AIR POLLUTION

AIR AND POLLUTION

A thin layer of air surrounding the earth, the atmosphere, makes life as we know it possible. The atmosphere is a mixture of gases, solid particles, water and other vapors. The major components of "pure" air are nitrogen, oxygen, carbon dioxide, and water. Carbon dioxide and water are essential to photosynthesis, the process by which plants use sunlight to produce their own food. Oxygen, nitrogen and water are necessary for the respiration, growth and development of plants, animals and most other living organisms.

Other substances are also present in air as a result of both natural processes and human activities: pollen, fungal spores, bacteria and viruses, dust particles, microscopic rock fragments, and chemical compounds such as ozone and ash. All of these and many other chemicals may be added to air as a result of activities such as agriculture, manufacturing, power generation, and residential living.

Substances and their proportions in the air vary over time and from place to place. This can be due to meteorological factors such as wind, temperature, humidity, and precipitation, and to varying geological and geographical conditions. For example, dust and other particles may fall to earth with rain; ozone levels increase after a thunderstorm; and pollen levels are high in the northeast in spring when the forest trees flower.

Air is regarded as polluted when contaminant levels either individually or in combination are known to or may adversely affect the health, safety and well-being of humans or the environment. Pesticides from agricultural spraying can cause health problems both as gases and when they are washed into water supplies. Radiation from serious nuclear power plant malfunctions or detonations can cause radiation sickness or death. High pollen counts produce severe discomfort in sensitive individuals.

Sulfur and nitrogen dioxides and carbon monoxide are produced by combustion processes such as forest fires and the burning of fuel in factories, homes and vehicles. Sulfur and nitrogen dioxides from factory emissions produce acid rain which damages plants and increases the acidity of lakes, leading to the death of fish and other aquatic organisms. High concentrations of carbon monoxide from motor vehicle emissions may be fatal to humans.

INDOOR AIR

As a result of dispersion and air movement the air inside a structure will contain all of the components of the ambient or surrounding air. Ambient air enters buildings through ventilation, which is the process of adding air

to or removing air from a space, by either natural or mechanical methods. Ventilation of a building and entry of ambient air can occur through open doors or windows, gaps or cracks in the building skin, as well as through mechanical ventilation systems.

Indoor air may also contain substances generated by occupants or their activities, objects in the building, or the structure itself. Occupants generate airborne substances through food preparation, fuel burning for heat or cooking, tobacco smoking, hair sprays, cleaning products, paints, and the occupants' own shed skin cells and hair. Contaminants may be produced by carpeting, plywood, concrete and pets. The air of an office might additionally contain chemicals from copying machines, pesticide spray residues, and air conditioning coolants. A factory may have particles and fumes from its manufacturing processes.

Since free passage of air is limited, substances emitted inside a building tend to accumulate. As a result indoor air may contain concentrations of some compounds which are greater than the ambient air. For example, an Environmental Protection Agency (EPA) study has shown that organic contaminants can occur indoors at up to 10 times the outdoor levels found in either industrial or rural areas. The level of a given substance tends to increase in winter when windows are kept shut. Pollutant levels are also higher in structures with poor ventilation and in settings where greater amounts of a source exist.

Results of time budget analyses show that the bulk of most people's time is spent indoors. In addition, studies in the United States, Canada and Europe indicate the greatest exposure to many pollutants occurs inside buildings (1). Thus it is important to determine which substances people are being exposed to indoors, the sources of these substances, and the effects which they have on human health, individually and in combination.

Asbestos

Products containing fibers of the mineral asbestos have been widely used in acoustical and thermal insulating material including pipe insulation, and in concrete, spackling compounds, ceiling and floor tiles and ventilation systems. Over 85% of the asbestos found in buildings is immobilized within some type of binding material. Release of high concentrations of fibers occurs when such material is disturbed, either accidentally or during maintenance, renovation, or removal. Exposure to asbestos fibers has been linked to lung and gastrointestinal cancer and other diseases. For example, prolonged exposure to high levels of asbestos can lead to asbestosis, a chronic lung problem typified by shortness of breath and extensive lung fibrosis. Skin irritation may also occur (8, 11).

Tobacco Smoke

Several thousand compounds have been identified in tobacco smoke including respirable particulates, nicotine, polycyclic aromatic hydrocarbons, nitrogen dioxide, and acrolein (11, 13). There is increasing evidence that exposure to other people's tobacco smoke affects respiratory health (11). It may cause irritation, coughing, sore throats and sneezing (14). Prolonged exposure to carbon monoxide and nitrogen dioxide may lead to chronic respiratory problems (5). An association has been found between eye irritation and carbon monoxide from the combustion of fuel or tobacco (4).

Although around 33% of adults smoke cigarettes regularly, a much higher percentage of children are routinely exposed. For example, 76% of children in a middle income community surveyed in St. Louis lived in homes with one or more smokers (15). Increased respiratory problems have been documented for infants in homes where parents smoke (16). Passive exposure to tobacco smoke increases the risk of lung cancer (17).

The presence of tobacco smoke may enhance the effect of both formaldehyde and radon (18). Burning tobacco produces formaldehyde, which will add to concentrations from other sources, and also produces particulates to which the radioactive radon decay compounds may attach and be inhaled.

Pesticides

Pesticides may be used indoors to kill roaches, ants, silverfish, wasps and hornets, and other insects and small invertebrates. Pesticides are often applied as sprays, or as liquids which evaporate and linger in the air. Vapors from outdoor applications especially on lawns or around foundations may enter indoor air. Airborne pesticides may be inhaled or settle on water or food and be ingested. Pesticides contain a variety of toxic compounds which have been identified as toxins, carcinogens and mutagens. Health problems may be associated not only with the active ingredients of pesticides but with inert compounds also contained in the products.

Biologicals

Inhalation of microbes, such as bacteria and viruses, which have been exhaled by other people and sometimes animals, is a primary route of transmission of most acute respiratory infections (19). Higher incidences of colds and flu during the colder parts of the year are probably the result of decreased ventilation inside buildings when windows and doors are kept shut. Serious disease and even death may result from biological contaminants in indoor air. Tuberculosis, measles, smallpox, and staphylococci are transmitted by ventilation systems in schools and hospitals. Bacterial aerosols are incubated in toilets, ice machines, and carpets and are distributed by humidifiers and cooling equipment (20, 21). Microbial hazards may be the most hazardous and also the most preventable category of indoor pollutants in supersealed buildings (3).

Allergens can lead to considerable acute respiratory discomfort; long term exposure can cause chronic conditions. Indoor allergens include pollen, molds and other fungi, hair, insect parts, and chemical additives. Pollen comes from cut flowers or flowering plants. Molds and other fungi grow on surfaces in warm, dark, damp areas, especially bathrooms and basements, and are generally more prevalent in summer. Insects enter a structure and die in light fixtures or by windows where they dehydrate and fall apart. Insects living indoors are found in food areas, dust and vents. They produce eggs, cocoons and webs, and shed skins and feces which can enter the air. Pets and people shed hair and dead skin cells. Dust mites, present in all indoor environments, feed on such debris from both humans and pets. These mites thrive in humid conditions and their feces are an allergen that can cause asthma (22, 7).

Combustion Products

The products of fuel burning for heating and cooking contain numerous hazardous compounds including formaldehyde, nitrogen and sulfur dioxides and carbon monoxide. These can accumulate indoors when stoves, heaters or furnaces are unvented or improperly vented, or when exhaust from internal combustion engines infiltrates buildings through open windows and ventilation systems. Carbon monoxide, for example, is fatal in high concentrations, and in low concentrations may lead to problems such as impaired vision and brain function (5).

Volatile Organic Compounds

Formaldehyde is one of many VOCs which are contaminants of indoor air. People emit acetone, alcohols, butyric and other acids. Certain polycyclic aromatic hydrocarbons are produced in the combustion of tobacco, wood and kerosene. Pesticides can release organophosphates or chlorinated hydrocarbons. In addition chlorinated compounds, acetone, ammonia, toluene, and benzene are found in paints, lacquers, varnishes, cleaning materials, and personal care products. The EPA studied 20 VOCs found in the indoor air to which 600 residents of New Jersey, North Carolina, North Dakota and California were exposed. Outdoor air measurements of VOC levels were recorded as well as

VOC levels in the residents' drinking water and breath. Exposure to 11 of the 20 VOCs was consistently higher indoors than outdoors. (10). Health risks of many of these have yet to be fully investigated (11). However, many are known or suspected human carcinogens, mutagens or teratogens. Some people (referred to as "chemies") are extremely sensitive to fumes from household products such as aerosols, paints, and cleaning products.

HISTORY AND SCOPE OF INDOOR AIR POLLUTION

History

Indoor air quality may have been an issue as long ago as the discovery of fire. Ceilings of prehistoric caves show layers of soot (14). Prior to the availability of techniques to measure substances in indoor air, the only efforts at control involved dilution of unpleasant odors. This approach arose from a concern that smelly places must be unhealthful. In reality foul smelling substances are not necessarily dangerous. Odorless or pleasant smelling substances, on the other hand, can be very hazardous (23).

Certain indoor air problems have been known in industrialized countries for decades. Minimum ventilation requirements began to appear in model building codes in the 1940s and 1950s. The widespread use of these codes, however, did not come about until the 1970s.

A school building code has been in place in Massachusetts since the 1940's. Massachusetts adopted its first comprehensive building code for new construction in 1975. Some town codes prior to this date included ventilation considerations; others did not. As a result a sizable portion of our existing building stock has been constructed without any minimum acceptable standards of ventilation. Moreover, ventilation requirements in codes are intended to reduce the transmission of airborne respirable diseases, to control odor and humidity, to provide combustion air for heating and to remove cooking by-products. Nonetheless, past concern about air quality was directed toward outdoor and industrial settings. Government efforts at quality control were similarly concentrated in these areas. Over the past several decades some new indoor pollutants have been discovered such as radon and new concern has arisen about the health effects of others, including formaldehyde and pesticide residues.

Buildings consume 1/3 of America's energy (6). When energy costs increased in the early 1970s there was a move toward decreased fuel consumption and alternative fuels. Both efforts have led to indoor air quality problems. Increased building insulation and general tightening of construction to prevent leakage are conservation measures initiated in response to high fuel costs. High levels of insulation are placed in ceilings, floors and walls: practices which tighten houses by blocking routes by which warm air escapes. Ducts of heating, ventilating and air conditioning (HVAC) systems are sealed

and heavily insulated (24). Thermal doors and windows are used (5). Heavily insulated shades and curtains are pulled across windows at night.

Tighter buildings have lower air exchange rates; that is, the rate at which the air in a room or structure is replaced by "new" air is decreased. At the same time, large numbers of wood stoves have been installed since the 1970s. Lack of regulation has led to high concentrations of emission pollutants such as carbon monoxide, nitrogen dioxide, and formaldehyde when stoves are not properly vented (14). Other improperly vented heating and cooking appliances and synthetic building materials pour contaminants into indoor air. Radon and tobacco smoke accumulate. The decrease in air exchange rates means that contaminants entering a room are being removed at a lower rate and thus reaching higher concentration levels.

Problem Scope

The extent of indoor air quality problems is unknown. Elevated radon levels have been found in structures in most states (25, 26); up to 1/3 of Americans smoke cigarettes (8); formaldehyde and asbestos are still found in products in many buildings; pesticides are widely used; biological contaminants are ubiquitous and alternative fuels are growing in use.

The indoor air quality issue is complex. While many sources of pollution have been identified, information on levels found in structures, particularly homes, is available for only a few pollutants. Little is known about long term health effects of exposure to either single pollutants or to several simultaneously. Since indoor air may contain many different substances, questions regarding health risk must be asked for each substance and for combinations of these substances. That is, if both substance "A" and substance "B" are present, they may react. "A" might cause more severe effects in the presence of "B" than when it occurs alone. If levels vary widely it will be difficult to predict the risk of illness to any one individual (5).

Sick Building Syndrome

In recent years people who live and work indoors have reported many symptoms of physical discomfort such as fatigue, headache, sore throat, nausea and eye irritation. Such symptoms have appeared most frequently in tightly constructed buildings, often without operable windows. This has led to the phrases sick building syndrome (SBS) and tight building syndrome (TBS) (27).

SBS has been known since World War I. The first SBS study was not published until 1948 in England (3). According to Woods, Janssen, et. al., a building is considered "sick" when the following conditions exist:

(1) Symptoms such as those described above occur in more than 20% of the building occupants. The 20% arises from a thermal comfort ventilation

rate guideline established by the American Society of Heating, Refrigeration and Air Conditioning Engineers (ASHRAE) which specifies a certain number of cubic feet of fresh air per minute (cfm) per occupant. The guideline is based on the cfm which will be comfortable for 80% of the visitors to a room (27).

(2) The cause of the problem is not recognizable (27). In some cases there is no obvious source of contaminants. In others the source is known but the means by which it is transmitted from its point of origin to the area where complaints occur is not known (28).

(3) Most people with discomfort indicate relief almost immediately after leaving the building. An SBS problem is considered to be resolved when the cause is identified, modifications are made and complaints cease (27).

Causes of SBS may be either physical or psychosocial. A physical cause is almost always found, for example, poor ventilation or contaminated air. The ventilation system is often the major problem either because it is not achieving an appropriate air exchange rate or the system itself is a source of contamination. An adequate air exchange rate may not be achieved if the ventilation system is not used and properly designed and maintained, or if the building use has changed or expanded without altering the ventilation system accordingly (29). Some sources of contamination which have been found include: outside pollutants such as carbon monoxide, nitrogen dioxide from automobile exhaust which is drawn into the building, microbes and fungi which incubate in ventilation ducts, and fumes or smoke circulating from other parts of a building. Psychophysical factors such as stress or job dissatisfaction are often found in addition to physical causes (27).

Perhaps the best known case of SBS is the one which led to the discovery of Legionnaire's disease. In 1976, 221 American Legionnaires attending a convention in Philadelphia became sick and 29 of them died. The cause of their illness was determined to be a disease associated with bacteria in the cooling towers of their hotel. The bacteria were named Legionella which, along with other harmful bacteria, have since been found in other buildings (3).

Contamination in other cases has been traced to water towers, ventilation systems and air ducts. Bacteria such as Legionella, staphylococcus, streptococcus, and fungi such as Penicillium can breed and enter the air circulating in the building (29).

Numerous other occurences of SBS are known. In 1984 two dozen employees at The New York Times became sick. The cause was found to be birds' nests and feces located near the air conditioning equipment which contaminated the indoor air (3).

In an elementary school outside Washington, D.C., four teachers lost a total of 350 days of work due to problems in the building. The school was built in a low area with no provision for drainage. Water leaks were found in the walls and dampness led to mold growth on books and materials (3).

People working at the veterinary school at the University of Florida in Gainesville became sick in 1986. The cause was a faulty ventilation system. Thus far the university has spent 1 million dollars appropriated by the Florida legislature to purchase modular buildings to temporarily house staff. The cost of repairing the ventilation system is 3.4 million dollars, 1/3 the cost of constructing the building in 1978 (29).

SBS may be widespread. The Fireman's Fund Insurance Company found that 1/3 of the 48 buildings it investigated around the United States posed indoor air pollution hazards and subsequently, in 1983 established an SBS laboratory and consulting service (24, 30). By 1986 the National Institute for Occupational Safety and Health (NIOSH) had investigated complaints in 450 public and private office buildings. Problems were found with fabrics, microbes, and contaminants from both inside and outside. Fifty percent of the buildings had inadequate ventilation (31).

MEASUREMENT AND MONITORING OF INDOOR AIR QUALITY

Pollutant Identification and Concentration

In order to evaluate excessive indoor air pollution and its health effects it is important to identify which pollutants are present in a room or building, and to determine how the levels of each vary with time. Monitors are available for particulates and a few gases such as radon, formaldehyde, nitrogen dioxide, sulfur dioxide, and carbon monoxide (32). Specific pollutant monitors can be installed for a period of time and the results analyzed in a laboratory.

In situations such as SBS, the source of contamination may be unknown, and testing for a broad spectrum of possible pollutants may be required. Analytical methods used are complex, time-consuming, expensive and require expertise and specialized equipment (5).

Total Individual Exposure

The effect of airborne pollutants on a given individual may be cumulative. The concentration and types of pollutants vary over time, within a room, from room to room within a building, between buildings, and from indoors to outdoors. An individual divides his or her time among many places, both indoors and out. Since the effects of pollutants often depend upon the total exposure an individual receives, it is important to monitor the individual through his daily activities.

Methods to determine an individual's total exposure to indoor airborne pollutants may be subjective or objective and represent either an assessment

of existing conditions or a reconstruction of past exposure. Determination of the amount of exposure may be via anecdote, history, activity diary, or personal monitoring.

"Anecdotal information" or recollection relies on a person's memory of past conditions and may provide valuable clues to a present physical condition. Anecdotal information however is often vague and inaccurate. There also may be inaccuracies because some contaminants are colorless and odorless and some like radon decay products and microorganisms may be below perception thresholds (14). A "history" is often more specific and may consist of medical records, and data such as the number of smokers in the home at various times, occupational background, and living conditions.

A personal, or activity, diary provides a record of daily activities such as the amount of time spent indoors and outdoors and the characteristics of the indoor environments. If this is coupled with measurement of the pollutant levels in the areas where time is spent, an exposure profile can be developed.

Because of the variability of pollutant levels over time and from place to place and, since most people do not remain in one place all the time, the only means of quantitatively assessing total exposure is "personal sampling." With this technique a person carries a monitor while going about his usual activities.

A number of limitations exist with personal sampling in addition to the lack of availability of monitors for many gases: analysis can be costly and time-consuming; many industries will not allow samplers on their premises so the people sampled may not be representative of the general population; monitors may be cumbersome or annoying so some persons may not wear them; and wearing them may modify behavior.

RISK ASSESSMENT FOR EXPOSURE TO INDOOR AIR POLLUTANTS

Risk assessment is a tool developed by epidemiologists and toxicologists to determine the probability of a given health effect arising in a population from exposure to a given chemical substance or mixture of substances. A National Academy of Sciences (NAS) model, presented to the Commission by John Graham of the Harvard School of Public Health, uses three factors - hazard identification, dose response and exposure assessment - to develop a risk or probability profile for a substance (2, 33). The following discussion is based on this model.

The first component, hazard identification, is a determination of whether the substance affects human health and, if so, in what ways. For a substance to be deemed a hazard, a causal link must be found between the substance and one or more health effects.

Once the hazardous nature has been established, it is important to know the likelihood that various concentrations will produce health effect and how

the severity of these effects varies as concentration increases. This is dose response, the second component of risk assessment.

Exposure assessment, the third component, seeks to determine whether a hazardous substance actually occurs at health threatening levels.

Hazard Identification

A substance is hazardous if it presents a health risk; that is, its presence leads to discomfort, disability, disease or death. Evidence to support the hazardous nature of a substance may come from structured human or animal studies, exposed occupational groups, histories including medical records, anecdotal information, or personal diaries. Each information source has strengths and limitations.

Studies of laboratory animals permit testing of sufficient numbers of individuals to produce statistically meaningful results. Short life spans of small animals permit lifelong and multigenerational studies yielding results within a reasonable period of time. However, results of animal tests may not always be extrapolated to humans because of metabolic differences among species and the use of test dosages which may be higher than those to which humans are exposed (34).

Occupational studies of workers exposed to a hazardous substance often provide the first evidence of the hazardous nature of a substance. Levels of the hazardous substance are not always known and other substances may also be present, making it difficult to pinpoint the cause of a health problem.

Medical records may provide an individual's occupational history and lifestyle, including time spent in various environments, and personal habits such as smoking or diet. Conclusions based on medical records of individuals cannot be extended to the general population. This is because patients do not represent a random cross-section of a population, but rather those individuals who have developed problems.

Anecdotes may help illuminate a problem, yet it is sometimes difficult to know their accuracy. Anecdotal information may suffer from bias on the part of the patient, or forgetfulness of events or situations which occurred in the past. Personal diaries may be incomplete if an individual forgets to keep the diary or eliminates certain events or situations because they are not considered relevant or the person is embarassed to report them.

While each individual study or type of study may leave some uncertainty as to the hazardous nature of a substance, as more studies accumulate a clearer picture emerges. The hazardous nature of indoor air pollutants being studied by the Commission has already been established.

Dose Response

The hazard posed by a substance may vary with concentration (dose) and with total exposure, or a dose over a period of time. Dose response studies are used to assess the health effects of different doses or total exposures to a pollutant. Such studies can indicate when a health effect appears and how its intensity varies as dose or total exposure increases. Dose response studies are generally undertaken once a substance has been shown or is strongly suspected to be a hazard and may utilize any of the methods described above.

The dose response relationship may take one of several forms. It is linear if the magnitude of a health effect increases in direct proportion to either dose or total exposure (Fig. 2-1). A sigmoid relationship occurs if at certain low doses or exposure times there is no health effect (Fig. 2-2). At some threshold an effect occurs and then increases as in the linear model. There may also be a point where increased doses or exposures no longer increase the magnitude of the effect; for instance, if a certain dose of a substance causes death, a higher dose cannot cause more death. In addition to linear and sigmoid curves, other relationships may also occur.

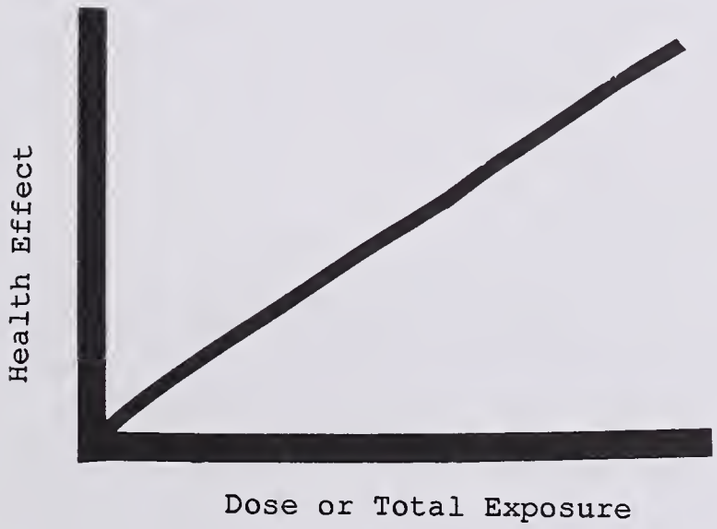


Figure 2-1. Linear Relationship of Health Effect to Dose or Total Exposure

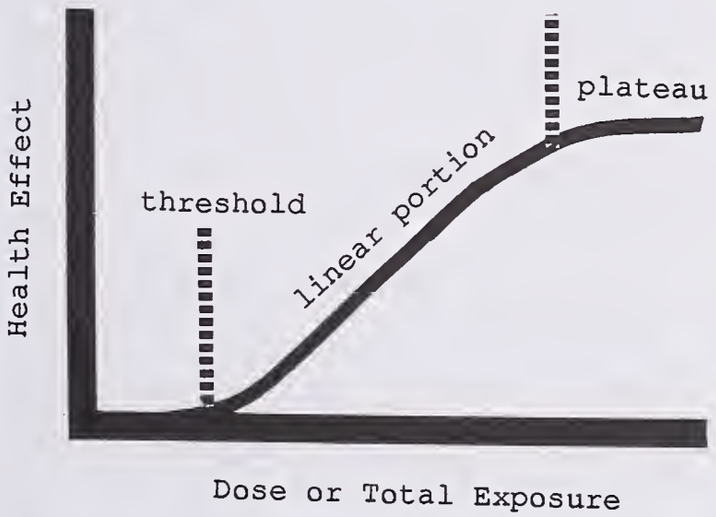


Figure 2-2. Sigmoid Relationship of Health Effect to Dose or Total Exposure

For some substances hazardous effects are known only for high doses. Studies are needed to determine the effects of lower doses and whether a health effect threshold exists.

Exposure Assessment

Once a substance is established as hazardous and its dose response relationship is known, exposure assessment may be used to determine whether health-threatening levels exist. To assess exposure to indoor air pollutants it is important to establish not only whether health-threatening levels occur, but also how much variation there is from structure to structure or from time to time in the same structure, how many structures are affected and which individuals are being exposed to which levels. Extensive studies may be needed to develop a full profile of exposure.

Risk Characterization

Knowledge that a substance presents a hazard, along with dose-response information and exposure assessment, allows development of a profile which characterizes the nature and magnitude of the human risk.

The scheme outlined above is a framework to assess a problem. In an ideal situation the nature of a hazard, all its possible health effects, its interactive effects with other substances, its dose-response behavior, and the full extent of the population's exposure to the problem would be known. Such perfect information is rarely available, yet policy decisions are often required in the face of scientific uncertainty and incomplete data bases.

Massachusetts Methodology

Risk assessment techniques for ambient toxics in Massachusetts were developed jointly by the Office of Research and Standards and the Division of Air Quality Control in the Department of Environmental Quality Engineering (DEQE). Their goal was to develop health-based recommended ambient air levels. This Chemical Health Effects Assessment Methodology uses three factors in assessing health effects: weight of the evidence, potency, and severity of the effect.

Weight of the evidence is a qualitative assessment of the strength of the data; potency is a quantitative assessment of the dose response; and severity of effect is used to distinguish minor effects, such as irritation, from more serious irreversible effects. These factors are used to assess each of four categories of health effects resulting from exposure to a hazardous substance: acute/chronic toxicity, carcinogenicity, mutagenicity, and developmental and reproductive toxicity.

recovering up to 80% of the heat that would otherwise be lost. Distribution of fresh air throughout a structure can be achieved by the use of separate ductwork supplying and removing air at remote locations or by circulation through existing warm air heating or air conditioning systems.

A third method of actively ventilating is to pressurize a building with outdoor air, forcing diluted indoor air out through leaks and openings. This is the typical method employed in most commercial buildings when outdoor air is drawn directly into return air ducts through adjustable dampers, displacing indoor air which would otherwise be recirculated.

For all ventilation systems, maximum efficiency will occur when exhausting from locations of maximum contamination. When pollutants are evenly mixed throughout a space and the source rate is constant, the concentration of airborne pollutants will be inversely proportional to the ventilation rate; that is, doubling the ventilation rate will halve the concentration. This relationship is known as the dilution principle (Fig. 2-3). Active ventilation systems provide continuous ventilation to which passive ventilation may add but not subtract. They can also provide for occupant control to suit occupant sensitivity and to handle variable sources and source strengths.

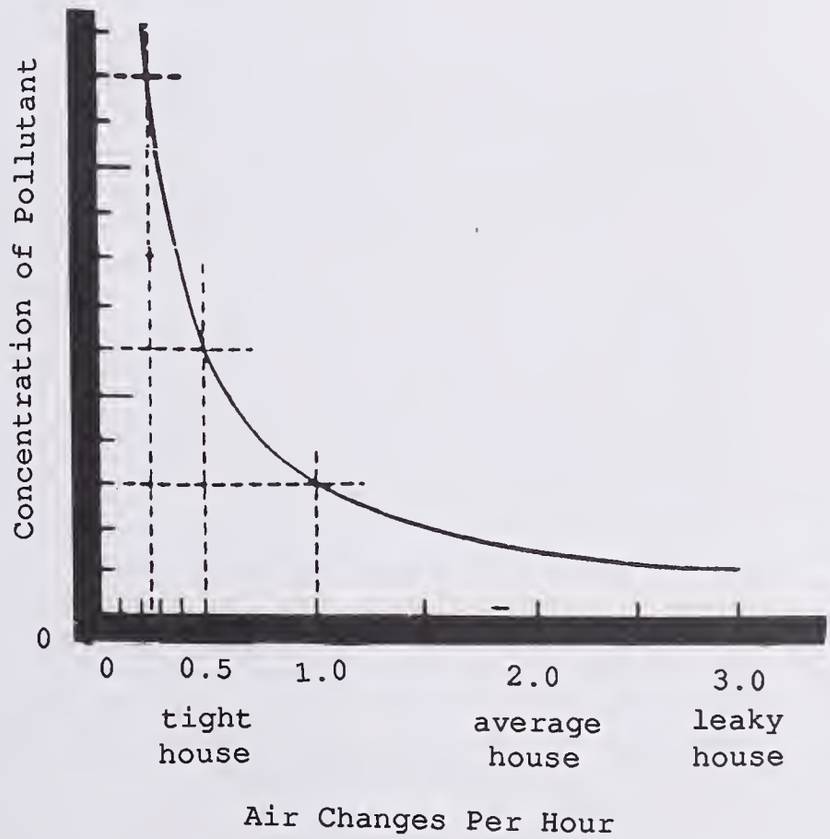


Figure 2-3. The Dilution Principle
Concentration of Pollutant is Inversely Proportional to Ventilation Rate.

An existing ventilation system which is inadequate because of design flaws, poor maintenance or expanded use of a building is often associated with poor indoor air quality. Mitigation can often require redesign or maintenance. In cases where the outdoor air ventilation provision of an HVAC system is simply not being used, the remedy is obvious.

By measuring the carbon dioxide concentration in an occupied space and comparing levels with outdoor air, the effectiveness of a ventilation system can be assessed. Ventilation rates are commonly determined by measuring air pressure differences with a pitot tube, or by anemometers or tracer gas techniques. A tracer gas, such as sulfur hexafluoride, can be released near a suspected contamination source and its movement through the building can be assessed (28).

Air Cleaning

Various types of filtration systems which remove airborne contaminants are available. Included among these are electrostatic air cleaners, media filters, activated carbon, and air washing systems. In electrostatic precipitators (or electronic air cleaners) particles pass over electrically charged plates or wire grids, become charged, and deposit onto oppositely charged plates or grids. Media filters range in effectiveness from coarse filters such as those used in forced air furnaces to fine high efficiency particle arrester systems. Filtration is primarily applicable to particulates and not appropriate for removal of gases.

Some types of air cleaning technologies such as activated carbon and air washing systems which can remove gaseous contaminants are available. These are generally impractical or cost-prohibitive for comfort applications. Activated carbon media need to be changed regularly and the effectiveness of electrostatic precipitators depends on regular maintenance.

INDOOR AIR POLICY

Policy Decisions

Policy provides a framework for government to address a problem such as indoor air pollution. The process of policy development involves complex interactions among affected groups with diverse viewpoints (34). A common way to build a foundation for policy is through a consensus panel which draws conclusions based on available evidence. Such a panel might include, for example, representatives from government, business or industry, consumer groups and technical experts.

Spengler and Sexton present a straightforward approach for developing informed policy decisions regarding indoor air pollution. They discuss five

The Department of Public Health (DPH) uses the three major steps outlined by the National Academy of Sciences (NAS) and discussed above in preparing risk assessments: hazard identification; dose-response assessment; and exposure assessment.

INDOOR AIR POLLUTION MITIGATION

There are two basic strategies to mitigate unacceptable levels of airborne pollutants in structures: addressing the source of the pollution and addressing the levels of contaminants in the air. These may be referred to as "source control" and "removal" respectively.

Source Control

Sources of pollution can either be man-made or exist in nature. When practical, source control is the preferred mitigation procedure for minimizing the level of an airborne pollutant since it prevents or reduces the ability of the source to become an indoor contaminant. Total elimination or avoidance of a source is a permanent solution. Other control techniques will reduce contamination levels by reducing source quantities or by sealing procedures.

Source control measures are pollutant specific and there are a number of current examples. These include the use of low formaldehyde-emitting construction materials or substitution of non-emitting materials. Cigarette smoking can be banned in public places or in homes; foundations can be sealed to reduce radon entry; insulation other than asbestos can be used; paints and solvents can be stored outside of an occupied space and safer cleaning and health care products can be substituted.

Another example of source control is treatment of biologically-contaminated equipment and materials such as medical and laboratory supplies and facilities, carpets, and furniture. The most appropriate solution to use, however, is a matter of controversy. Chlorine bleach is effective but because it is caustic to eyes, nose and skin, caution must be used. Ammonia is also not always effective. Glutar-aldehyde based products are used in hospitals, doctors' offices, laboratories and industrial facilities on everything from equipment to walls. Some of these burn the eyes and nose and have a strong, lingering odor. More acceptable forms are being developed such as a mixture of sodium chlorite and water.

Preventive measures against microbial contamination include maintaining water temperature above 120 degrees Fahrenheit, keeping relative humidity between 40-60%, using steam humidifiers and avoiding standing water in HVAC systems (3). When an HVAC system is the contamination source, proper maintenance may be the solution, or redesign may be required to prevent birds from nesting in the cooling tower or to create accessibility to ducts where organisms are breeding.

Removal

Contaminants may be removed from a building and replaced with outdoor air by means of passive or active ventilation, or they may be removed from the indoor air by using air cleaning devices. If contaminants are generated at a faster rate than they are removed, the concentrations in the indoor air will increase. Conversely, removal at a faster rate than generation will result in a decrease in concentration of those contaminants. Ventilation is effective for removal of all indoor pollutants to a greater or lesser extent depending upon the pollutant type (35).

Passive Ventilation

Structures without active ventilation rely on open doors and windows or other intended or unintended openings in the structure for air exchange. Passive ventilation refers to air exchanged through these openings by natural forces. The rate at which air is passively exchanged depends on many factors, including size and number of openings, wind speed, temperature difference between indoor and outdoor air and occupant living patterns. Generally, maximum ventilation rates will occur during extremes of wind velocity and outdoor air temperature. Negative indoor air pressures can be induced by these factors and in turn increase the rate of radon entry into homes.

Most of the 82 million homes in the United States have no mechanical ventilation and current residential building practices rely principally on passive ventilation. The energy-conserving tight construction of recent years has reduced the amount of air flow and therefore the rate of pollutant removal. A typical new house in the northwestern United States experiences 0.6 ach while duplexes, apartments and mobile homes range from 0.3 to 0.6 ach. This may not always be adequate since present information indicates high levels of certain pollutants in many of these structures. Older homes generally have about 1.0 ach and newer homes around 0.5 ach (31).

Active Ventilation

Active ventilation occurs when electro-mechanical devices such as blowers are used to achieve an exchange of indoor and outdoor air. Active "exhaust-only" systems rely on unintended openings in a structure for outdoor air supply. Typically, the most effective system of this type for ventilation of an entire structure would provide one or more points of active, continuous exhaust, connected by ductwork to remote spaces having openings for entry of outdoor air. Spot ventilation provides for intermittent removal of certain pollutants at their source, such as range hoods and bathroom fans.

Other ventilation systems actively exhaust and supply fresh air to a structure. Such systems provide a higher assurance of achieving designed ventilation rates. In addition, because air is being mechanically exhausted and supplied, the counter-flowing air can be ducted through air-to-air heat exchangers for the purpose of conserving energy. In the winter, most of the heat in the exhaust stream can be transferred to the colder incoming air,

aspects: problem definition, health risk estimation, mitigation measures assessment, identification and resolution of public policy issues, and decisions regarding appropriate government response (36).

Problem Definition

Indoor air pollution arises from a complex of contaminants from many different sources. The concentration of each pollutant in the air of a room can vary independently due to differences in the rate the pollutant is being generated and the rate it is being removed from the air. Each pollutant can lead to one or more health problems especially since certain combinations of pollutants may interact. Little is known about such synergistic effects. Severity of an effect can depend on the concentration of the pollutants, the length of exposure and the sensitivity of an individual.

Thus a clear understanding of indoor air pollution requires identification of pollutants, sources of pollution, factors which affect the rate of pollutant entry, factors which dilute contaminants, the range of concentrations which are found in indoor air, how exposure varies with peoples' daily activity patterns, and the types of health effects which may result (36).

Until recently most indoor air quality research focused on problem definition. Areas which have been most heavily researched include: nitrogen dioxide and carbon monoxide from unvented combustion (mostly gas stoves); formaldehyde from UFFI, building materials, and furnishings; and concentrations of airborne particles such as those present in tobacco smoke. Research is now increasing with environmental tobacco smoke, radon decay products, VOCs and particulate phase organics. Although microbes may be an important source of health problems, some of which are fatal, levels of bacteria, viruses and fungi are seldom measured (36).

Risk Assessment

A complete risk profile for an indoor air pollutant would include the range of health effects which occur from the presence of a given pollutant or mixture of pollutants, how the effects differ as the level of pollutant rises, whether immediate or long term exposure is more important to the development of the health problems, what interactive effects occur among pollutants, how many people are being exposed, the levels of exposure they are receiving, and how their susceptibility differs. When all of these are known, it is possible to predict the chances of an individual developing a given health effect. Good risk profiles are essential in order to devise control strategies for reducing risk.

Mitigation Measures Assessment

Mitigation or relief of indoor air pollution ideally means decreasing the concentrations of pollutants in the air to levels where no long or short term health effects occur. The general types of mitigation include: source control through removal; substitution or modification of the contaminating substance; removal of contaminants through ventilation or air cleaning; and changing the living patterns of the occupants. Under current technology some techniques

are more appropriate than others for a given contaminant and some contaminants are handled with a combination of methods. Behavior modification depends on awareness of a problem by the occupants or owners of a building and on a willingness to alter personal or work habits. Awareness can be enhanced with public education, product testing and labeling, and clarification of legal rights and responsibilities (36).

Policy Issues

A key component of informed policy decisions is a clear understanding of the issues. Policy questions must be identified and then resolved before indoor air quality problems can be addressed. Policy questions pertaining to indoor air pollution include:

(1) Does government's role differ in public versus private structures?
(2) Should voluntary and involuntary risks be considered separately, such as cigarette smoking versus exposure to the smoke of others?
(3) Should long term and short term exposures to indoor pollutants be addressed differently?
(4) Should the same degree of protection be considered for the general population as opposed to infants, the ill, the elderly, or other particularly sensitive individuals?
(5) How should responsibility be divided among individuals, building owners and operators, architects, developers, contractors, manufacturers and government?
(6) Is government action appropriate and if so what type and level of action is required?
(7) At which level of government should authority and responsibility be vested?
(8) What are the cost-benefit tradeoffs between energy conservation and indoor air quality? (36)

Scientists vary in their views of when action should be taken regarding health policies. Some advocate action based on slim evidence by appealing to emotional factors. Others will not act without solid proof from controlled experiments and often find themselves in the company of special interest groups. Some do not want to take a stand or equivocate by placing undue conditions or qualifications on their findings. They become hypercritical of all studies (34).

Government Response

While scientists and health officials recognize that contaminated indoor air is a major exposure route for many pollutants, government must decide how to protect public health while faced with incomplete and sometimes conflicting information (36). Government responses to indoor air pollution problems might include legislation, regulation, guidelines, suggestions, education, or no action. Some potential specific responses include:

(1) Encouraging research to fill data gaps central to decision-making;
(2) Treating indoor air quality as a personal choice issue and emphasizing education and labeling;

(3) Developing economic incentives or disincentives to encourage adequate indoor air quality;
(4) Promoting voluntary industry codes and guidelines;
(5) Defining legal responsibilities and liabilities;
(6) Establishing guidelines to assist individuals, professionals and regulatory officials;
(7) Promulgating rules and regulations including design standards, such as building codes and minimum ventilation requirements; emission standards, such as limits for appliances, consumer products and building materials; and indoor air quality standards, such as maximum allowable concentrations for certain contaminants;
(8) Taking no action, based on little public health risk or unfavorable cost-benefit ratios (36).

Appropriate responses to indoor air problems depend on resolution of the policy issues. If air of private residences is considered a matter of personal choice (as was decided with saccharin or cigarettes), then the appropriate response may be education or product labeling. If offices or schools are considered areas where the individual has little or no control over air quality, then regulation may be appropriate (36).

Constraints on Government Action

Federal government actions regarding indoor air pollution are currently constrained by a number of factors:
(1) inadequate research funding;
(2) lack of firm scientific basis for action;
(3) lack of consistency in approach or centralization of authority for indoor air quality issues;
(4) reluctance of federal officials to act without clear statutory authority;
(5) reluctance of regulators to become involved in controversy over whether private spaces should be regulated; and
(6) fear by some regulatory and environmental entities that acknowledging indoor air problems will weaken the case for outdoor standards (36).

ASHRAE

ASHRAE has been active in promoting ventilation guidelines which lead to acceptable indoor air quality. ASHRAE feels government has a role in sponsoring research on indoor air quality and its health consequences and in contaminant control technology. Its rationale is that professional societies, manufacturing or trade associations and industries lack the comprehensive interest, resources and competence to insure well-funded systematic research programs (6).

ASHRAE has made a number of proposals, including the following:
(1) Increase in government funded health effects research. The purpose of

health effects research should be to determine physiological and toxicological effects of specific contaminants. This can be achieved through clinical exposure and animal studies; occupational health effects research; prospective epidemiological studies following populations over time with environmental and physiological measurements; and identification of sensitive individuals.
(2) National inventory of indoor air contaminants. This would identify populations at risk. The inventory should include air exchange rates, sources and resulting concentrations, and models of interactions among pollutants, air exchange rates and energy consumption.
(3) Continuous evaluation of information. As data accumulates indoor air pollution issues should be continuously re-evaluated to consider guidelines and ventilation standards established by government in cooperation with professional societies.
(4) Address acute problems. Government should provide education, legal, financial, and technical help immediately to deal with acute problems.
(5) Incorporate ASHRAE guidelines into state and local codes. ASHRAE Standard 62-1981, Ventilation for Acceptable Air Quality, is suggested as the most comprehensive ventilation guideline balancing energy conservation and health.
(6) Examine policy options to reduce indoor air pollution levels. To achieve this, look at market forces, source control, manufacturing controls, property and operational regulations, and establishment and enforcement of standards.
(7) Involve private sector. The private sector, including professional societies, should be involved in improving products and performance standards for instruments, materials, equipment and HVAC systems and in enhancing professional practices of society members.
(8) Assist professional societies. Professional societies should be aided in disseminating information to other professionals and the public. This can be achieved, for example, through joint sponsorship of research, publications, and conferences (6).

INDOOR AIR QUALITY: FEDERAL AND NATIONWIDE INITIATIVES

Authority

Past concern about air quality focused on outdoor or industrial settings and federal and state programs concentrate on these areas (36). The EPA is the lead federal agency for air pollution control. Through the 1970 Clean Air Act Congress gave EPA authority over ambient air. The term "ambient," however, has been interpreted to mean outdoor air (1, 36). Thus indoor air pollution is not considered to be covered by the Clean Air Act and no single federal agency is responsible for controlling indoor air quality (37).

Similarly the Clean Air Act amendments of 1977 did not specifically charge EPA with indoor air quality, but it is the agency with indoor air quality closest to its central mission and is thus regarded as the lead agency by Congress (1). The General Accounting Office also defines the term ambient

as outdoor air, but acknowledges the need for indoor air quality protection and recommended that EPA be given this authority (36).

Title IV of the 1986 Superfund Amendments and Reauthorization Act (SARA) gave the EPA a formal mandate to conduct research and disseminate information on indoor air pollution (36).

There are a number of other federal statutes under which EPA might control indoor air quality. These include:
(1) The Toxic Substances Control Act (TSCA), which is aimed at toxic air pollutants. EPA used TSCA to require asbestos removal from schools and is considering regulatory action regarding formaldehyde exposure;
(2) The Federal Insecticide, Fungicide and Rodenticide Act (FIFRA) applies to pesticides used indoors;
(3) The Uranium Mill Tailings Radiation Control Act (UMTRCA) applies to tailings used for landfill in residential areas or in dwelling construction. EPA has developed guidelines for radon concentrations in homes in high risk areas;
(4) The Safe Drinking Water Act (SDWA) could be used where drinking water comes from radon-emitting soils or rocks; and
(5) The Comprehensive Environmental Response, Compensation, and Liability Act (CERCLA) or Superfund might be used where volatile organics or radionuclides from hazardous waste sites can travel through soil to buildings (36).

Sixteen federal agencies are responsible for various aspects of indoor air quality (37). No single federal agency has authority over the non-workplace. There is a definite precedent for authority inside buildings through building codes and fire ordinances (51). In addition to EPA, those agencies with powers over aspects of non-industrial indoor air quality are: the Occupational Safety and Health Administration (OSHA), the Department of Energy (DOE), the Bonneville Power Administration (BPA), the Consumer Product Safety Commission (CPSC), the Department of Housing and Urban Development (HUD), and the Federal Trade Commission (FTC) (36).

OSHA and NIOSH are equipped to monitor for biohazards. NIOSH, which was created as the research arm of OSHA, published guidelines for builders and building operators in the mid 1970s. These included criteria for a recommended standard for occupational exposure to carbon monoxide, a study of occupational health in hospitals focusing on indoor environmental elements, and criteria dealing with exposure to fiberglass (3). NIOSH has investigated over 450 sick buildings (31).

DOE, responsible for energy conservation programs in new buildings and residences, has funded research to improve measurement techniques and assess health effects from radon, airborne particulates and organic vapors (36). DOE has studied the relationship among indoor air quality, energy conservation and radon, including health effects, epidemiological and building science studies. EPA and DOE are developing a memorandum of understanding to coordinate their programs. DOE will have primary responsibility for basic research, with its principal focus on health effects. EPA will handle applied research, technical studies, and operational programs which deal with the states and the

private sector (1). DOE, however, lacks specific authority to address health effects. DOE's only mandate is to save energy; therefore, DOE can only address preventing deterioration of air quality beyond existing levels.

CPSC ensures products do not create unreasonable health effects, and has banned spackling compounds with asbestos, proposed a ban on UFFI (which was overturned in court), and funded emission studies for unvented combustion appliances (36). CPSC has done work on emissions including combustion products, formaldehyde and methylene chloride (1).

HUD develops building standards for projects it funds and material standards for mobile homes. In high radium areas in South Dakota and Montana, indoor radon concentrations must be below a certain minimum as a requirement for HUD financing. HUD has refused approval of Federal Housing Administration (FHA) loans for home construction on reclaimed Florida phosphate lands because of high radon potential. HUD has also developed regulations limiting formaldehyde emissions from plywood and particle board (36).

The FTC ensures the truth, accuracy and usefulness of consumer advertising, and recently charged two room air cleaner makers, who claimed their products remove pollutants including tobacco smoke, with false advertising (36).

The Department of Health and Human Services (DHHS) also has responsibility for indoor air quality and regards EPA as the lead agency for indoor air quality. DHHS principally funds and coordinates a Harvard six cities study of health effects from both indoor and outdoor pollutants (1).

DOE, CPSC, DHHS and EPA are represented on the interagency Committee on Indoor Air Quality (CIAQ) and conduct, support and administer indoor air quality research (1). Currently the Committee is assessing federal indoor air research programs.

EPA Activities

After a presidential veto of a similar bill, two million dollars was appropriated in 1984 and again in 1985 for EPA research on indoor air quality, representing only 3% of its overall air budget (36). Title IV of the 1986 Superfund Amendments and Reauthorization Act (SARA) for the first time gave EPA a formal mandate to conduct research and disseminate information on indoor air pollution. EPA's initial work under SARA has concentrated on the relationship of indoor versus outdoor levels of criteria pollutants (1).

In a report to Congress required under Title IV of SARA, EPA announced its intention to take a dual approach to indoor air pollution by concentrating both on individual products and pollutants and on the structures where they are found. Its program emphasizes identification, characterization and ranking of indoor air pollutants and their health effects, along with assessment and implementation of mitigation strategies. It will identify high risk pollutants and determine exposures and health risks of various populations.

It will also look at total exposure to indoor air pollutants and develop mitigation techniques for multiple pollutants using building design and management techniques (1).

EPA will assess federal risk reduction mechanisms and take action using existing statutes such as SDWA, TSCA, and FIFRA or through other agencies with regulatory power from Congress. It will emphasize information dissemination and ultimately increase the abilities of state and local governments and the private sector to identify and solve immediate health problems and reduce risks (1). EPA will also encourage research projects to improve sampling techniques (38).

EPA has addressed a number of indoor chemicals through TSCA and FIFRA. The statutes allow EPA to obtain information from manufacturers and processors when any substance they use potentially presents unreasonable risk to health or the environment. This is based on risk-benefit analysis (1).

EPA has issued the Asbestos Worker Protection Rule which protects public employees not covered by OSHA from asbestos exposure during abatement projects. EPA has also proposed an Asbestos Ban and Phase Down Rule to reduce future uses and exposures. The Asbestos Hazard Emergency Response Act (AHERA) passed in 1986 requires schools to inspect, prepare management plans and take action if friable asbestos is present (1).

EPA banned pentachlorophenol as a preservative on log-homes in 1986, and both pentachlorophenol and creosote as indoor preservatives for most uses. Phaseout of lindane in residences was issued in 1983, with total prohibition in 1986. EPA is currently assessing chlordane, heptachlor, and aldrin/dieldrin (1). Risk assessment results for formaldehyde were released in 1987.

EPA's air research program includes an approach called Total Exposure Assessment Methodolgy which uses personal and ambient monitors to determine human exposure. VOCs for example were found to exceed outdoor levels by up to 500%. Via the Integrated Air Cancer Program EPA developed instruments and methods to characterize airborne carcinogens. Studies include effects on children of smoking parents (1).

EPA has built a testing chamber to study pollutant emissions from building materials and consumer products and thus far has analyzed paints, floor waxes, and other substances. EPA is currently working on standardization of emissions testing procedures and developing a model to estimate exposure from volatilization of chemicals in tap water. EPA has set water emission standards under the SDWA and is considering using this statute to establish maximum levels for VOCs since they vaporize in hot water (1, 3).

EPA participated in a study with the National Academy of Sciences (NAS) leading to the Surgeon General's report on environmental tobacco smoke and health (1).

Recently, the Region 1 EPA office in Boston has established a workgroup on indoor air quality problems in New England. This workgroup, the New

England Indoor Air Quality Workgroup (NEIAQW), is comprised of representatives from state and federal agencies that address indoor air issues in the region. It is scheduled to meet about three times a year to discuss technical and policy issues of concern to the states.

Standards, Regulations and Guidelines

World-wide outdoor air quality standards do not consider indoor exposures. From the 1940s to the 1970s indoor exposures were based on ambient air and emissions (39). Little attention was paid to indoor air except in industrial settings because it was assumed that buildings sheltered people from outdoor pollutants (40). It is now known that levels in houses sometimes exceed outdoor standards. These standards cannot be readily applied to indoor levels since people are exposed over longer periods indoors. Outdoor standards also do not always take into account the heightened susceptibility of the very young, ill or elderly (35).

EPA is responsible for establishing and enforcing National Ambient Air Quality Standards (NAAQS) (36). Through its authority under the federal Clean Air Act, EPA has established standards for a few pollutants in the outdoor air designed to "protect the public health...with an adequate margin of safety" (42). Standards define the concentration of a pollutant to which most people can be exposed for a given period of time without adverse health effects. Standard concentrations are normally listed in either parts per million (ppm) of air or micrograms per cubic meter of air (5).

A number of states and organizations have developed standards or guidelines for indoor pollution levels. These apply mostly to workplace or public buildings. As yet there are no air quality standards which apply to all residences in the United States (5).

In the United States the tendency is to address comfort rather than health. OSHA regulates concentrations of pollutants in the workplace and enforces industrial "consensus" standards designed to protect employees from "material impairment of health or function" (36, 43). ASHRAE publishes ventilation system design and operational guidelines as well as minimum outside air requirements.

In 1977 ASHRAE published indoor ventilation guidelines for buildings constructed in the eight preceding years. In 1981 these were updated. Its Ventilation for Acceptable Indoor Air Quality Guideline requires higher air exchange rates in areas where tobacco smoking is allowed (3).

In public, commercial and office buildings the current ASHRAE guidelines are 5 cfm/person of air in nonsmoking areas and 25 cfm/person in smoking areas. These guidelines fail to recognize pollution from newer materials and equipment, however, and are being revised upward to most likely 15 cfm for nonsmoking and 60 cfm for smoking areas (31). OSHA is promulgating new indoor air quality standards based largely on NIOSH and ASHRAE research (3).

ASHRAE has recommended as a guideline for residential, office or retail spaces that the exposure of the general public should not exceed 1/10 of the industrial occupational standard for a given pollutant (43). The recommendation was intended to provide a comfortable environment. ASHRAE points out that this recommendation is no guarantee of protecting health, particularly for sensitive or sensitizable persons. Following the recommendation prevents short term effects for many pollutants but does not address the effects of long term low level exposure (44).

A variety of regulatory agencies in other countries as well as federal, state, and local governments have required ventilation rates on the order of 0.5 ach in dwellings. Europe and Canada are far ahead of the United States in addressing indoor air problems. The Canadian Standards Association requires 0.5 ach of mechanical ventilation in all mobile homes. The National Building Code of Canada now calls for 0.5 ach of mechanical ventilation in all new dwelling units. Canada's R-2000 Super Energy Efficient Homes Program requires a mechanical ventilation capability of 0.5 ach. Sweden and France have adopted 0.5 ach of mechanical ventilation requirements for all new construction. The California Energy Commission requires 0.7 ach of mechanical ventilation for the tightest of California's new residential buildings. Others, including South Dakota, Wisconsin, and the Northwest Power Planning Council, have adopted or are proposing requirements for mechanical ventilation which, combined with the assumed natural infiltration rate, provide 0.5 ach in residences (45).

Congress passed the Northwest Regional Power Act in 1980 directing the four northwest states to develop Model Conservation Standards in order to capture all the energy savings "cost-effective to the region" and "economically feasible" for the consumer. The Model Conservation Standards require 0.6 ach. Tight energy-efficient homes require air to air heat exchangers to achieve this standard (46).

INDOOR AIR QUALITY: STATE, MASSACHUSETTS AND LOCAL INITIATIVES

Other States

There has been some activity at the state and local level to cope with indoor air quality problems. Health departments receive most of the complaints and calls for information. Thirty-two states including Massachusetts have a person or program responsible for evaluating exposures to at least one indoor pollutant; 29 have some non-industrial formaldehyde assessment program. Many state and local governments have anti-smoking ordinances to reduce exposure to passive tobacco smoke (36).

California, the only state with a comprehensive indoor air quality program, has banned the sale and use of unvented combustion space heaters in dwellings (36). California OSHA has mandated owners of structures in which people work to operate ventilation systems during all working hours and operate and maintain such systems to local building standards. Owners must

also keep precise maintenance and inspection records. California OSHA took this step after receiving 350 reports a year from office workers with problems ranging from headaches to severe allergic reactions (30).

In 1987 the Minnesota Department of Health began formulating a protocol for determining "whole" indoor air conditioning of a building rather than addressing individual pollutants (46). Minnesota, and Wisconsin have developed formaldehyde standards for new mobile homes and a law in effect for eleven years restricts smoking in a variety of indoor places including restaurants, retail stores, and offices (36).

At least 12 states require nonsmoking areas in restaurants. In 1986 Rhode Island passed a statute requiring employers to make reasonable accommodation for nonsmoking employees, especially those particularly sensitive to tobacco smoke. This could be achieved by partitioning or by increasing ventilation, but the employer was not obligated to spend money to do so (47).

Massachusetts

In Massachusetts indoor air policies and programs are fragmentary and scattered among various agencies, similar to the situation at the federal level. Presently Massachusetts has programs in the DEQE, DPH, and the Department of Labor and Industries (DLI) to address certain aspects of radon, asbestos and formaldehyde. DEQE's Divison of Air Quality Control can address some indoor pollutants when the source occurs outdoors. DLI has issued guidelines for investigation of "bad air" complaints. The protocol includes checking for chemical exposure sources, reviewing the symptoms of the occupants and testing for certain compounds including formaldehyde and carbon monoxide. Criteria are recommended by the Division of Occupational Hygiene for ach and percent of fresh air.

Since the energy crisis of the mid 1970s, DPH has received numerous complaints of poor indoor air quality, particularly in schools and buildings occupied by the general public. The ventilation requirements in the Massachusetts Building Code for schools had been reduced from 10 cfm to 5 cfm per occupant. The reduced fresh air ventilation requirement resulted in increased respiratory infections and frequent complaints of discomfort. DPH was successful in revising the code in late 1986 to again require 10 cfm fresh outside air per occupant.

In 1979, DPH banned the further use of UFFI in Massachusetts after corroborating numerous homeowner complaints of health effects attributable to formaldehyde exposure. A residential formaldehyde action level of 0.1 ppm was established. An air testing and evaluation program was implemented in 1986 to provide remedial relief to homeowners with formaldehyde levels above 0.1 ppm or with documented formaldehyde related health problems. Approximately 7,500 homes in the state are thought to have been insulated with UFFI; 3,000 of these homes have been air tested to determine present indoor formaldehyde levels. The average level to date in a UFFI home is 0.04 ppm with 2-3% of the homes tested indicating levels above 0.1 ppm.

A broad 1987 statute restricts smoking to designated areas in restaurants with a seating capacity of 75 or more. No barriers need be provided. In addition public institutions of higher learning must provide a certain number of dormitory rooms for nonsmokers. Nursing homes must provide nonsmoking sections in common areas and employees may not smoke in patient care areas. No smoking is permitted in courthouses, schools, colleges, museums, libraries, trains, airplanes or airport waiting areas, the waiting areas of certain health care facilities, or day care centers except in designated areas. However, designated areas need only be provided if there is sufficient space for nonsmokers.

There are local regulations in over 50 Massachusetts communities requiring nonsmoking areas in restaurants. A 1981 DPH poll showed 66% of residents, including 52% of the smokers, favor restrictions on smoking in restaurants. A 1983 Gallup poll indicated 91% of nonsmokers and 86% of smokers prefer either separate sections or a total ban on smoking in restaurants.

Chapter 3: RADON

THE ELEMENT RADON

Radon is a chemical element which occurs as a colorless, odorless gas. While it does not react to form compounds, radon is radioactive. All radioactive elements undergo spontaneous decay which produces both new elements and radiation in the form of alpha particles, beta particles or gamma rays. If the elements produced are also radioactive, further decay will occur until stable, non-radioactive elements are formed (48,49).

Radon 222 is part of a chain created as uranium 238 decays, and is the immediate decay product of radium (Fig.3-1). Radon in turn undergoes decay to isotopes of polonium, bismuth and lead which are referred to as "radon progeny" (Fig. 3-2) (50). Decay of progeny continues until stable lead 206 is formed.

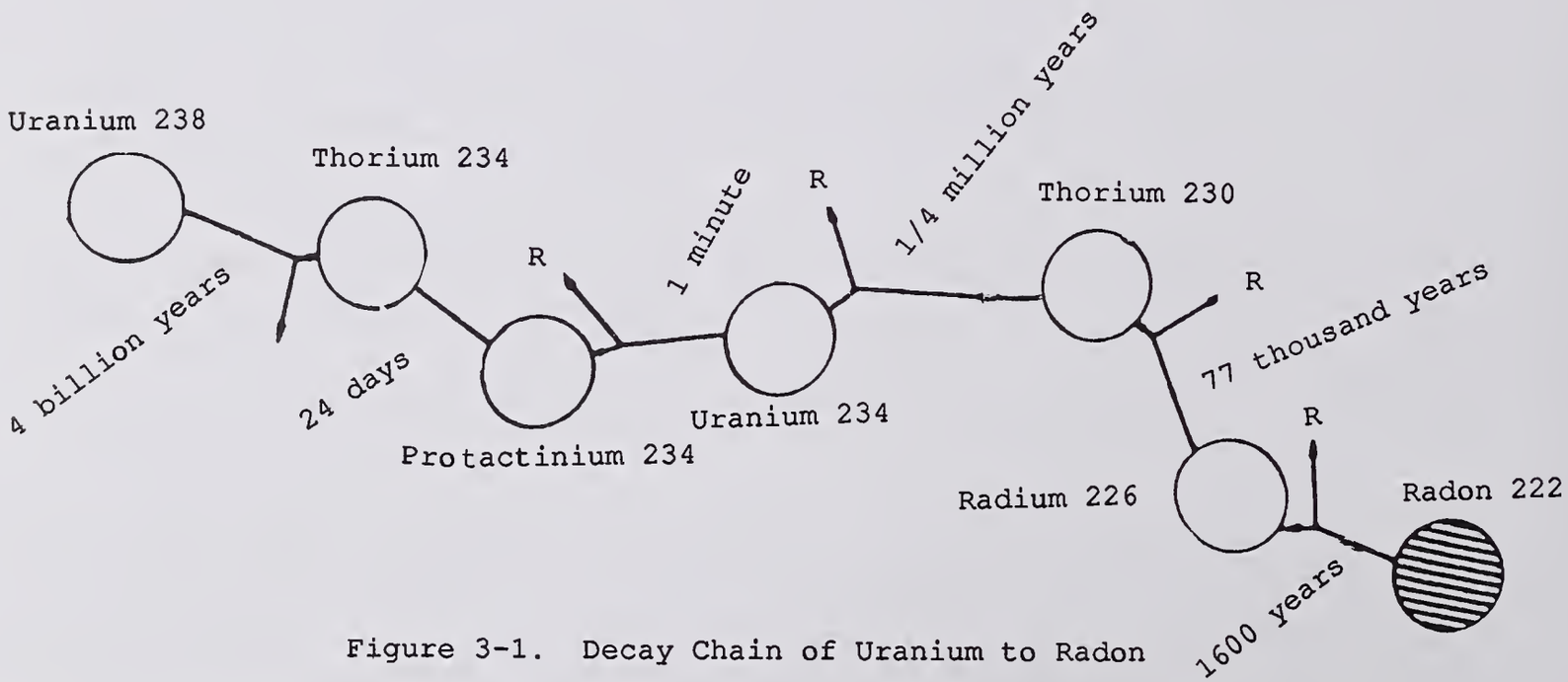


Figure 3-1. Decay Chain of Uranium to Radon

(Source: University of Maine and Maine Department of Public Health, 1983)

Each radioactive element decays at a specific rate called its half-life, or the time required for 1/2 of the atoms in a sample to decay. Radon produced today is from the uranium present when the earth was formed. The half-life of uranium is very long. Radon, however, decays with a half-life of 3.8 days and its progeny in a matter of minutes or seconds (Fig. 3-2). Thus radon gas and radon progeny are sources of imminent radiation (50).

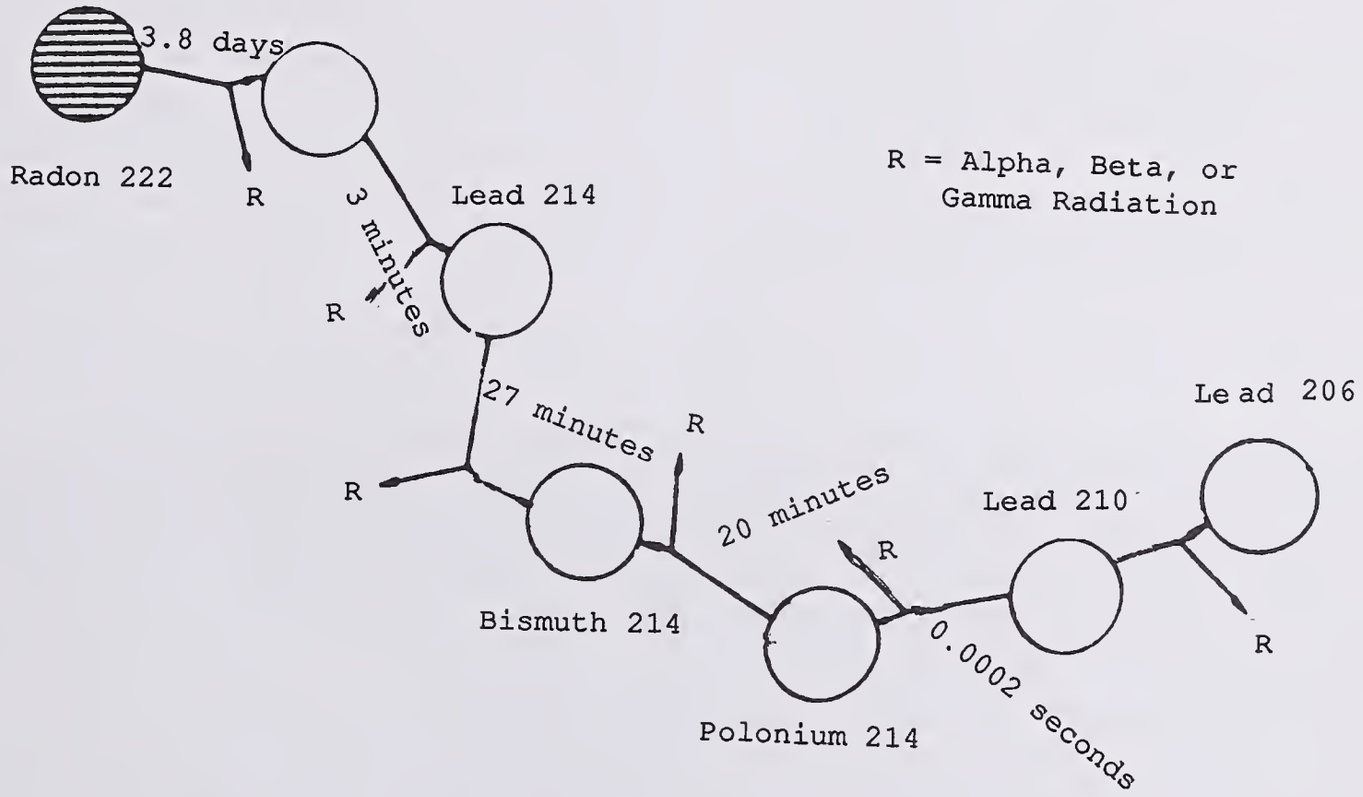


Figure 3-2. Decay Chain of Radon to Lead

(Source: University of Maine and Maine Department of Human Services, 1983)

Uranium and other radon precursors as well as radon itself are almost ubiquitous components of rocks and soil in the earth's crust (9). Uranium is 40 times as common as gold and almost as abundant as tin (39). Radon gas escapes to the atmosphere from uranium-bearing surface rock and the soil formed as such rock decomposes. Radon from subsurface rock may also reach the surface as a gas via fissures in the rock or dissolved in groundwater.

Although they represent minor sources, fossil fuels such as oil, coal and natural gas may contain uranium and therefore radon. Activities or technologies which redistribute uranium such as mining, well drilling and burning of fossil fuels, bring radon to the earth's surface.

Radon in coal can be released through mining and burning, in disposal areas, in runoff from fly ash, or from construction materials made of fly ash. Higher levels appear in coal mined in the western U.S. If gas production sites are located near radon-bearing rock, natural gas used in unvented appliances such as kitchen stoves and space heaters may emit radon. Propane, which is a by-product of natural gas processing, may also produce radon (48). Radon is also found in the by-products of phosphate and uranium mining and in construction materials such as brick or concrete, if they are made from uranium-bearing rock.

RADON AND PUBLIC HEALTH

Physiological Effects

While radon has no known immediate effects (51), exposure may increase risk of lung cancer and is a suspected cause of stomach cancer (49)(50). Decay of both radon and its chemically active progeny may produce damaging physiological effects in humans. Airborne radon and its progeny may contact the skin and, if inhaled, the nose, mouth, throat, esophagus, and lungs. Similarly, radon in water may contact the skin and decay or, if ingested, decay in the mouth, stomach, and intestines. Radon can pass through the wall of the gut, into the blood where its progeny can damage the stomach and other organs. Progeny, however, cannot pass through the gut.

Inhaled radiation can hit and damage molecules in living cells (50). Beta particles and gamma radiation tend to break molecules in only one place if at all. Alpha particles (which are actually fast moving helium nuclei) are the major source of health risk since they tend to cause more than one break in molecules they hit (48). Cells possess certain chemicals which can repair breaks. If a molecule has several breaks, there is a chance the pieces may be put back together incorrectly. If this occurs with a genetic molecule (DNA) it may reproduce itself incorrectly leading to a mutation (50). Many mutations are harmful and may cause the cells containing the DNA to function abnormally. Cells may also be killed.

In the case of radon, which is principally inhaled, the major concern is from radiation affecting the function of cells in the lungs. Radon progeny are radioactive solids which will "stick to" anything they come in contact with, including airborne particulates. A fraction of progeny do become attached. Both attached and unattached progeny may decay further, releasing radiation in the process (50). Radon progeny attached to particles and those existing as free ions or atoms have different likelihoods of reaching the lungs and depositing in different parts of the respiratory system (48)(49).

The attached fraction is mostly deposited in the pulmonary region of the lungs with little removed by the nose (52).

Radon and Lung Cancer

The illness contracted in the late 1800s by miners in Germany and Czechoslovakia was ultimately identified as lung cancer. Mortality was 75% and was greater for miners than for the masons and carpenters working the same mines. In 1924 radon was suggested as the cause (50).

Uranium and other miners exposed to high radon levels have significantly higher lung cancer rates than the general population. For example, of 4000 miners who worked in the 1940s and 1950s, there were 151 lung cancer deaths compared to the 16 which were expected in a comparable number of members of the general population (50). This increased lung cancer in miners eventually led to air quality standards and remedial measures in the mines.

Concentration and Total Exposure

The decay rate of radioactive material is measured in Curies (named for the discoverers of radium - Marie and Pierre Curie). A Curie is the amount of any nuclide which undergoes 3.7 x 10 exp10 radioactive disintegrations per second. A pico Curie is one-trillionth of a Curie (5). Radon concentration (or dose) is often measured in pico Curies per liter (pCi/1). One pCi represents decay of approximately 2 radon atoms per minute in a liter of air (51).

Radon progeny concentration is expressed in working levels. One working level (WL) was originally used to define the highest level of radon permitted in a workplace. Since alpha particles are the major cause of tissue damage, the definition of WL was subsequently adjusted to mean the amount of alpha energy potentially available in one liter of air for any combination of radon decay products. One WL equals 1.3 x 10 million electron volts of potential alpha energy per liter of air. It is estimated that 1 pCi/1 of radon will produce a progeny concentration of 0.005 WL under ideal conditions (51). One working level month (WLM) is the exposure for 1 month (or 170 hours or approximately 21 8-hour working days) to 1 WL (9).

A "safe" annual exposure was originally set at 12 WLM then reduced to 3. It should be noted that WLM is defined for a workplace. In speaking of WLM's for residences it is necessary to adjust for the actual number of hours per day a person is spending in the home, which is about four times as long as a miner works.

Prior to radon reducing control measures the air of mines averaged 4000 pCi/1. Areas away from the mines measured only around 1 pCi/1. Lung cancer thus came to be regarded as an occupational problem.

Mortality rates of the miners indicate a delay of 10 to 25 years between exposure and appearance of lung cancer (50). The rate of death in the miners was found to be proportional to the total radiation exposure, or the radiation level multiplied by the length of exposure (13). This suggests that total exposure may be an important variable in risk calculations.

While levels found in buildings are usually much lower than those levels found in mines, total exposures for people living or working in situations with even low radon levels can be considerable. Thus the identification of elevated levels of radon in buildings has raised concerns that such levels may increase risk of lung cancer.

Risk Characterization

Estimates of the risk of dying from lung cancer as a result of radon exposure are based on models which assume a dose over a period of time and take into account the fact that people only spend a portion of their time in the structure in question. For example, an EPA model assumes 75% of the time is spent at home for a lifetime of 70 years. As Table 3-1 shows at 4-20 pCi/1 1-5 out of every 100 people would be expected to die from lung cancer. At 20-200 pCi/1, 6-21 deaths would occur and at 200 pCi/1 44-77 deaths would occur. In the latter case if the same 100 persons lived only 10 years in a house with 200 pCi/1, only 14-42 deaths would result (49). All of these are very high risk factors. Governmental action is usually considered appropriate when the risk is 1 in 100,000. It should be noted that the EPA action level is somewhat arbitrary. Public health concern should not stop at 4 pC/1.

About 4 out of every 100 people die of lung cancer. The Surgeon General estimates about 85% of lung cancer deaths are due to smoking (49) and EPA's Science Advisory Board estimates that about 10% are due to radon (49). Radon could cause roughly 10,000 deaths per year. Radon in water may be responsible for about 1000 deaths each year (9). The risk of a smoker dying of lung cancer is increased considerably if the smoker is also exposed to elevated levels of radon (18).

It is useful to compare the risk of lung cancer death from radon exposure to that of smoking (Table 3-2). For example persons exposed to 4pCi/1 have 5 times the risk of a nonsmoker; 200 pCi/1 is 60 times the nonsmoker risk and equivalent to that of a 4 pack a day smoker (49).

The above estimates could be low. An NRC study released early in 1988 (18) suggests the lung cancer risk from lifelong exposure to radon is several times greater than earlier estimates.

Table 3-1 Radon Risk Assessment

pCi/l	WL	Estimated number of lung cancer deaths due to radon exposure (out of 100)	Comparable exposure levels	Comparable risk
200	1	44 —77	1000 times average outdoor level	More than 60 times non-smoker risk
				4 pack-a-day smoker
100	0.5	27 —63	100 times average indoor level	20,000 chest x-rays per year
40	0.2	12 —38		
20	0.1	6 —21	100 times average outdoor level	2 pack-a-day smoker
10	0.05	3 —12	10 times average indoor level	1 pack-a-day smoker
				5 times non-smoker risk
4	0.02	1 —5		200 chest x-rays per year
2	0.01	.7—3	10 times average outdoor level	
1	0.005	.3—1	Average indoor level	Non-smoker risk of dying from lung cancer
0.2	0.001	.1—.3	Average outdoor level	20 chest x-rays per year

Source: U.S. Environmental Protection Agency

RADON IN STRUCTURES: EARLY FINDINGS

Concern over radon inside structures arose in the late 1960s when elevated levels were found in Colorado homes built on uranium mine wastes. Subsequently high indoor radon levels were found in various parts of the country in buildings situated over debris from other industrial activities.

Still later high levels of radon were found in homes built over natural deposits of uranium-bearing rock or soil (49).

Colorado Mine Tailings

The mill tailings left when uranium ore is extracted from rock remain high in uranium. From 1953 to the mid-1960s the use of uranium mill tailings for construction was common in Colorado for landfill and backfill and in cement. This practice ended when the radon levels in these buildings were found to be higher than in structures not built on tailings (48).

Florida Phosphate Lands

Phosphate rock contains uranium, thorium and radium which are distributed with the products, by-products and wastes of phosphate mining. Phosphate used as fertilizer, phosphoric acid and elemental phosphorous may therefore contain radon (1). Phosphate containing radon which is used for crop fertilizers may be taken up by plants, be deposited on the soil, or run off and enter ground and surface waters, as well as expose agricultural workers. Phosphate was once used in the United States for wallboard and plaster. Waste gypsum, a phosphate by-product, is made into concrete blocks (48).

EPA initiated studies of reclaimed phosphate mines in Polk County Central Florida in 1975. Eighty percent of phosphate mining in the United States occurs as strip mining in central Florida. After an area was strip mined the land was reclaimed by mixing mining by-products with the overburden (material above the phosphate layer) from an adjacent mine plot. About 25,000 reclaimed acres existed in the area in 1975, with another 75,000 acres potentially becoming available within the following 30 years. (53).

Thirty percent of the reclaimed land contained residential structures, 8% commercial structures and the remaining was agricultural and park land in 1975. The soil levels of radium 226, which decays to radon, ranged from 1-50 pCi/gram. Readings were highly variable over short distances. WLs of 0.015 - 0.018 (30-36 pCi/1) were found in single family houses on mineralized and reclaimed soils compared to 0.003 WL on non-mineralized soils (53).

Other Findings

When the New Jersey State Department of Environmental Protection, at federal request, surveyed sites where hazardous materials might be found, 243 homes in 3 communities were found to have been built on contaminated soil believed to be from a former radium processing plant (51,54).

In 1979 the University of California at Berkeley found radon levels of 20 pCi/1 in an energy efficient house in Maryland. This exposure is equivalent

to that of a Three Mile Island accident in the neighborhood once a week or the maximum exposure allowed to radiation workers. Twenty pCi/l was among the highest readings ever found aside from structures built on radon contaminated industrial mine tailings.

Then in 1984 a worker at the Limerick nuclear plant in Pennsylvania kept setting off radiation alarms. His home was found to contain 2000 pCi/l of radon. Subsequent investigation showed that his house was built on the Reading Prong, a large geological formation extending into New York and New Jersey and containing some of the highest radon levels in the country. These levels are more significant than releases from any nuclear plant and have led to widespread interest in radon in indoor air (55). The Maryland house and subsequent radon findings on the Reading Prong were the first indications of radon in structures built on undisturbed substrate.

RADON MEASUREMENT AND MONITORING

Findings of elevated radon levels in buildings on both natural rock and soil and on ground disturbed by industrial activities prompted questions regarding the distribution of uranium laden rock and soil, whether factors in addition to uranium content of the substrate affect indoor randon levels, by which pathways enters a structure, and how widespread the radon problem might be. Answers to such questions depend in part upon accurate measurement of indoor radon levels. Since radon is colorless and odorless, and doesn't burn or glow, it is undetectable by the senses. Thus its presence can only be detected with monitoring devices.

Some monitors measure the concentration of radon gas. Other monitors measure radon progeny concentration, expressed as WL (49). There are 3 types of radon monitors: prompt, time integrating and continuous readout. Some commonly used monitors are described below.

Prompt Sampling Monitors

Prompt or grab sampling monitors are used to give "instantaneous" readings of the amount of radon in the air at any given time. Detectors usually are left in place for just 5-10 minutes (51,56).

The Kuznetz method of grab sampling was developed for use in mines. An air sample is drawn through a filter which collects air particles with attached radon progeny. The filter is then aged for about an hour and the sample counted with a radiation detector. Progeny concentration is determined from detector count rate via a calibration table (51).

Time Integrating Monitors

Time integrating monitors are left in place for a period of weeks or months, where they continously absorb radon. The total amount of radon is recorded in some fashion and calculated often through laboratory analysis. This total is divided by the length of time the detector was left in place to determine the average radon concentration during the period of monitoring. Charcoal canisters and alpha track detectors are commonly used time integrating monitors. A charcoal canister detector consists simply of a container of charcoal with holes in one end which is left in place for 3-7 days (5,49).

Alpha track detectors contain a piece of plastic on which alpha radiation from decaying radon leaves microscopic marks or "tracks". After exposure the plastic is developed and the tracks counted. The density of tracks is then converted to radon concentration via calibration and described as pCi/1 for the time of exposure (5,49,51,56).

Continuous Readout Monitors

Continuous readout monitors sample the air and register readings continuously. They can be used to assess the variability in radon level over time. Continuous monitors can also be used to provide both grab samples and integrated readings depending upon the measurement period.

Testing Protocol

The above instruments, if properly calibrated, measure accurately and precisely. However climatic and meteorological factors, temperature and air pressure vary daily and annually and indoor radon levels vary accordingly. In fact levels may vary by a factor of 10 over a day or two. Thus longer term measurements than a few minutes are desirable (56). The time integrating monitors are the most simple to use and the least costly. The two commercially, most popular and preferred radon detectors of this type are charcoal canisters and alpha track detectors.

EPA Screening and Followup Procedures

A three step radon detection procedure is recommended by EPA: a screening measurement, determination of need for further measurements based on the initial reading and followup measurements. The screening measurement should be made under standardized conditions so measurements from different buildings can be compared. Testing should be done at the lowest livable level of a structure such as a basement. Measurements should be made when all windows have been closed for 12 hours and the outdoor temperature is below 40 degrees Fahrenheit EPA's recommendations for subsequent action depend upon the

initial results (See Table 3-2). If the screening measurement is less than 4 pCi/1, the EPA action level, followup is probably not required but it should be kept in mind that single measurements are always subject to some error. For those homes with screening levels of 4-20 pCi/1, EPA recommends retesting and if a level in excess of 4 pCi/1 is confirmed, appropriate mitigation should be implemented. For those homes with screening levels in excess of 20 pCi/1, immediate mitigation is recommended. Followup readings should be made in each living level (49).

Table 3-2 EPA Recommended Response to Indoor Radon Levels

Radon Concentration	Radon Progeny Concentration	Response
pCi/l	WL	
<4	0.02	followup probably not required
4-20	0.02-0.1	followup with detector in place for 1 year or 1 week each season
21-200	0.1 -1.0	followup with detector in place for 3 months
>200	>1.0	immediate followup with detectors in place no more than 1 week and action taken to decrease radon level

(Source: Massachusetts Department of Public Health. "A Citizen's Guide to Radon: What it is and what to do about it," 1986)

Radon in Water

To analyze radon levels in well water, a water sample is collected from below the surface with a hypodermic needle and the sealed sample is counted with a scintillation counter (57).

FACTORS AFFECTING RADON LEVELS IN STRUCTURES

Factors which determine indoor radon levels include: the uranium content of local rock and soil, soil chracteristics such as permeability, pathways into the building, ventilation and the pressure differential between the structure and the environment, and the habits and activities of the occupants (9, 48, 53). It is difficult to predict how these variables will interact to affect radon levels in any given structure.

Uranium Content of Underlying Rock and Soil

The amount of uranium present in the substrate beneath a building is clearly a major factor in the concentration of indoor radon. Some of the highest levels found occur in areas which have high levels of uranium or other radon precursors in the native soil or rock.

Most rock and soil, and granite in particular, contain 1-4 ppm of uranium. Some phosphate strata in Florida reach 120 ppm (9). Acidic rock such as granite, often has higher concentrations, whereas less acidic rock, such as limestone or sandstone usually has lower concentrations of uranium.

EPA has mapped the distribution of uranium-rich soils. Such soils occur throughout much of New England; in the Reading Prong area of eastern Pennsylvania; northwestern New Jersey; and southern New York; along the eastern slopes of the Appalachians; in the phosphate rich soils of Florida; along the Georgia and Carolina coasts; and in other areas scattered throughout Wisconsin, Minnesota and west of the Rockies (58).

The U.S. Department of Energy's National Uranium Resource Evaluation Program has used airborne detectors to monitor gamma ray production from bismuth 214, a radon decay product, in order to assess the country's uranium resources. Monitoring bismuth will give an indication of the amount of radon which is being generated (55).

A joint radon survey by the Connecticut Department of Health Services and Environmental Protection found that geological formation was a significant predictor of radon levels in both indoor water and air. Homes over granite formations had significantly higher radon levels than the rest of the state and homes over sedimentary formations were significantly lower. The depth of

overburden correlated positively with radon in water; that is, water coming from deeper strata had higher levels of radon (58).

In late 1986 the New Jersey Department of Environmental Protection commissioned a study of radon in residences in an attempt to relate potential radon problems with geological features. Areas believed from earlier evidence to be radon prone were more heavily sampled. Averages of 8.1 and 8.2 pCi/1 were found in the two most northerly areas, including the uranium rich Reading Prong, whereas the statewide average was 5.4 pCi/1.

EPA's Generalized Bedrock Geologic Map of New England (Fig. 3-3), which emphasizes uranium endowment, indicates numerous regions of Massachusetts that are underlain by granitic rock types with moderate to high uranium content, including much of southeastern Massachusetts, Cape Cod and Cape Ann. About 20% of the state is underlain by gneisses which vary from low to high in uranium content (59).

Because of variation in uranium content and other soil characteristics, radon content can be highly variable over short distances. This was found in the reclaimed phosphate lands (53). In one study of rowhouses in Ontario, those with the highest level were adjacent to those with the lowest (50).

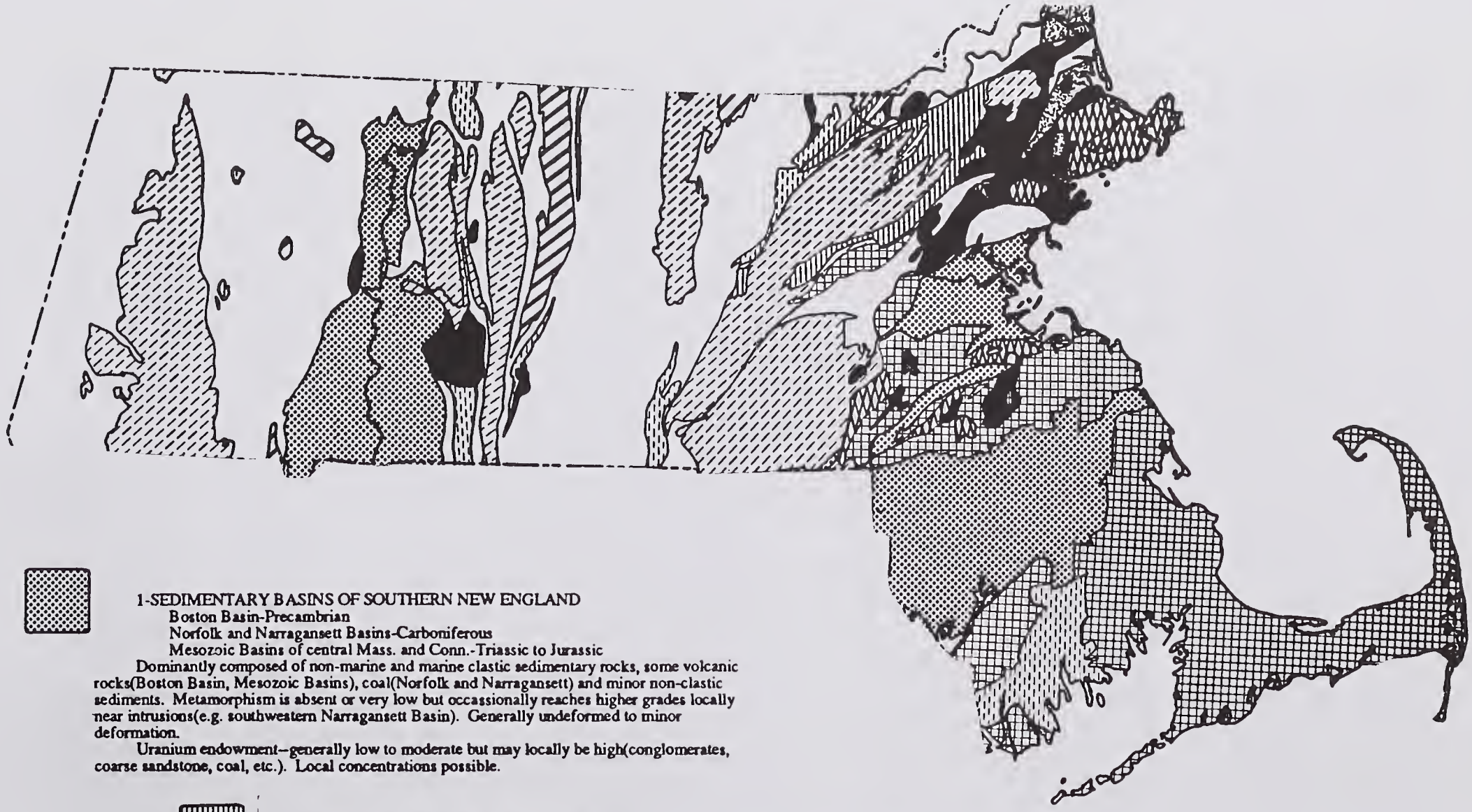
Other Soil Characteristics

Radon levels in a given soil are determined in major part by the soil composition, porosity, temperature and moisture, by the radon gas pressure in the soil, and by the pressure differentials between the soil and air. The rate of radon diffusion from soil to the air is affected by these same variables. Diffusion is reduced when the ground is cold or frozen, wet following heavy rain, under snow cover, and when barometric pressure is high. Review of nearly 1000 measurements indicates the average worldwide outdoor diffusion rate of radon from soil is 0.42 pCi/meter square/second. The range is from 0.006 to 1.4 pCi/meter square/second (9, 48, 51).

Concentrations of radon in outdoor air average about 0.1 pCi/1 or 0.0006 WL in the northern hemisphere. Concentrations vary with precipitation, barometric pressure, and time of day, in addition to local presence of uranium, soil structure and land use (51).

Radon reaches the atmosphere when it is emitted by soil, rock or water. A fraction of this may return to the ground. Rain may absorb some radon from the air as it passes through the troposhere where the highest atmospheric concentration occurs, and return it to soil and surface water.

Soil permeability is important to indoor radon levels. Radon formed in a bedrock containing high amounts of uranium may not reach the surface if the overlying soil is a relatively non-porous clay. Alternatively a rock quite low in uranium might lead to high indoor levels if overlain by a porous sand. Local variations in soil type over the same rock can lead to large differences in indoor radon levels.



1-SEDIMENTARY BASINS OF SOUTHERN NEW ENGLAND
Boston Basin-Precambrian
Norfolk and Narragansett Basins-Carboniferous
Mesozoic Basins of central Mass. and Conn.-Triassic to Jurassic

Dominantly composed of non-marine and marine clastic sedimentary rocks, some volcanic rocks(Boston Basin, Mesozoic Basins), coal(Norfolk and Narragansett) and minor non-clastic sediments. Metamorphism is absent or very low but occassionally reaches higher grades locally near intrusions(e.g. southwestern Narragansett Basin). Generally undeformed to minor deformation.

Uranium endowment--generally low to moderate but may locally be high(conglomerates, coarse sandstone, coal, etc.). Local concentrations possible.

2-BINARY OR TWO-MICA GRANITES
Various ages-generally Ordovician to Permian

Composed of intrusive igneous rocks of granitic composition with significant(>1%) amounts of muscovite mica. May contain small amounts of intrusive rocks of other types and compositions and xenoliths of country rock. Metamorphism absent to low grade(granitic rocks of medium to high grade are listed under granitic gneisses-8). Deformation is variable, many show well developed foliation, other are undeformed. Most are thin sheet-like bodies(<1km. in thickness) occurring in areas of medium to high grade metamorphic rock.

Uranium endowment--moderate to very high, local uranium concentrations.

3-GRANITES OF CALC-ALKALINE COMPOSITION
Various ages-Precambrian to Cretaceous

Composed of intrusive igneous rocks of granitic composition consisting of quartz, potassium feldspar, plagioclase, biotite and/or hornblende. Granites with significant muscovite are not included(see Binary Granites-2). Granites with large amounts of alkali feldspar, sodic amphiboles or other indicators of high alkali content are listed under Alkalic Plutonic Rocks-4.

Metamorphism is absent to low. Deformation is variable. Bodies of granite range from small sheet-like units to large batholith-size units of unknown thickness.

Uranium endowment--moderate to high, dispersed, usually no major local concentrations.

Figure 3-3. EPA Generalized Bedrock Map of New England

4-ALKALIC PLUTONIC ROCKS

Various ages-Ordovician to Cretaceous

Composed of intrusive igneous rocks rich in sodium and potassium indicated by large amounts of alkali feldspar or other alkali rich phases(e.g. riebeckite). Includes alkali feldspar granites, riebeckite granites, alkali feldspar quartz syenites, alkali feldspar syenites, quartz syenites, and syenites.

Metamorphism absent to low grade. Deformation absent to moderate. Generally small to moderate size batholiths and stocks.

Uranium endowment--moderate to very high, generally dispersed, occassional local concentrations.

5-PLUTONIC ROCKS OF INTERMEDIATE COMPOSITION

Various ages-Precambrian to Devonian

Plutonic igneous rocks of intermediate composition, including granodiorites, tonalites, monzonites and quartz monzonites.

Metamorphism absent to low grade(medium and high grade intermediate rocks are shown under intermediate and mafic gneisses-9). Deformation variable. May contain plutonic rocks of other compositions. Bodies are small to large stocks and sheet-like units. Sometimes associated with granitic gneisses.

Uranium endowment--low to moderate, dispersed.

6-MAFIC PLUTONIC ROCKS

Various ages-Precambrian to Cretaceous

Plutonic igneous rocks of mafic composition including gabbros, diorites, monzodiorites, artz monzodiorites, and quartz diorites. May contain small amounts of rocks of other mpositions. Metamorphism absent or low grade. Deformation variable but generally low. Bodies are small to moderate size stocks and sheet-like units. Usually associated with other plutonic rocks of intermediate to granitic composition.

Uranium endowment--very low to low, dispersed.

7-GNEISSES--GENERAL

Various ages-dominantly Precambrain and Ordovician

Rocks with moderate to well-developed foliation and gneissic texture. Compositions highly variable but generally derived from sedimentary rocks, but also includes some igneous rocks. This does not include stratified gneissic units except in a few instances where mappable at this scale. Also does not include gneisses of probable plutonic origin. Often associated with migmatites and plutonic rocks of intermediate to granitic compositions. Bodies are small to large units usually fault bounded or showing intrusive contacts. Generally in elongate bodies conforming to regional trends.

Metamorphism is medium to high grade. Deformation is moderate to intense. May represent uplifted or remobilized basement rocks.

Uranium endowment--variable, low to high, depends on rock composition and metamorphic processes. Locally may be very concentrated but not mappable at this scale.

8-GNEISSES OF GRANITIC COMPOSITION

Various ages-generally Precambrian and Ordovician

Gneisses of granitic composition and probable plutonic origin. Moderate to well developed foliation and gneissic texture. May include granites, granodiorites, migmatites and gneisses of other compositions. A few stratified units are shown but most are intrusive bodies or fault bounded. In general, they are slightly elongate and follow regional trends.

Metamorphism is medium to high grade. Deformation is moderate to intense. Probably represent deformed and metamorphosed intrusive bodies.

Uranium endowment--moderate to very high, generally dispersed but local concentrations possible.

9-GNEISSES OF INTERMEDIATE TO MAFIC COMPOSITION

Various ages-Precambrian to Devonian

Gneisses of intermediate to mafic composition, generally of plutonic origin. May contain plutonic and gneissic rocks of other compositions. Small to moderate size bodies, most are elongate and conform to regional trends.

Metamorphism is medium to high grade. Deformation is moderate to intense.

Uranium endowment--very low to moderate, generally dispersed.

10-ULTRAMAFIC ROCKS

Ages-Cambrian to Silurian

Rocks of ultramafic composition including serpentinites, norites, harzburgites, troctolites, etc. Generally small bodies moderately to strongly deformed. Metamorphism is variable. Some may represent fragments of oceanic crust.

Uranium endowment--very low

11-STRATIFIED METAMORPHIC ROCKS

Ages-Precambrian to Devonian

Metamorphic rocks consisting of sequences of marine and non-marine clastic and non-clastic sedimentary rocks, volcanic rocks of mafic to felsic composition and other rock types. In general, variations in lithology are too small to be mappable at this scale. Metamorphism and deformation are highly variable, but generally are lowest in northern Maine, and increase to high metamorphic grade and intense deformation in southwestern New England; although other areas of high grade metamorphism are found throughout New England. Bodies of plutonic rocks and gneisses too small to be mapped at this scale are also included.

Uranium endowment is highly variable and is dependent on rock type, metamorphic grade and the types of plutons in the vicinity. Local high uranium contents can occur. These variations can not, in general, be mapped at this scale.

Figure 3-3 (continued)

A study by the Regional Air Pollution Control Agency of Dayton, Ohio found the uranium content of Ohio soils to be low to average, but some are highly permeable to gas. Homes built on gas permeable substrates such as sand, gravel or fractured bedrock had higher radon readings (60).

Pathways

Pathways of radon into structures are determined in part by construction type. Features which allow freer passage of radon gas contribute to high indoor levels. A number of structural components affect indoor radon levels including basements, crawl spaces, sump holes, foundation types, tightness, and number of levels.

Higher radon readings have been associated with houses containing basements, crawl spaces, sump holes, and above average amounts of insulation in addition to certain soil factors. In the above Ohio study the average concentration in houses with basements was 8.4 pCi/1 compared with 4.1 pCi/1 in houses with slabs (EPA's sampling protocol involves testing which includes the lowest potentially habitable area of a house). Houses with crawl spaces averaged 10.3 pCi/1 compared to 3.5 pCi/1 in those without crawl spaces. This is possibly because there is seldom any barrier between the crawl space and the floor. Other studies have found houses with vented crawl spaces to have lower radon concentrations than those with slab-on-grade construction or basements. Houses with sump holes averaged 12.2 pCi/1 compared to 4.7 pCi/1 in those without. Sump holes go through the slab and are in direct contact with the substrate beneath (53, 60).

In a Connecticut study foundation type predicted radon levels, with higher readings in homes with cinder block foundations as opposed to poured concrete and cellars with dirt floors (60). Cinder blocks are more porous than concrete. Radon concentrations have been found to be lower in multilevel houses, in non-air conditioned houses (more ventilation), and in houses built without masonry (a radon source) (53).

One earth-encased concrete house in Beverly, Massachusetts was tested at the request of DOE which considered it an outstanding example of energy conservation techniques. Radon concentration was measured at 200 pCi/1, 50 times the EPA action level. Of the 4 components assessed as contributing to high radon levels - uranium content of the soil, porosity of the soil, pathways into the house, and ventilation - uranium content and soil porosity were not unusually high. When a ventilation system and heat exchanger were installed radon concentration dropped to 2 pCi/1 (61).

Ventilation and Pressure Differential

Ventilation rates are another factor affecting radon levels in structures. A simple test performed during a meeting of this Commission indicated a reading of 1.6 pCi/1 during the first hour when the ventilation

system was left off. Shortly after the system was turned on readings began to drop. By the end of the second hour the room contained only 0.6 pCi/1. The movement of outdoor air through a structure will dilute and remove radon gas thus lowering its concentration.

However, negative pressures created by mechanical or passive ventilation and indoor/outdoor air temperature differentials can create negative pressures which promote the entry of radon bearing soil gas into the structure.

Habits and Activities of Occupants

Activities of occupants which can increase indoor radon levels include heavy use of showers, washing machines, and any other tasks in which water is agitated. Concentrations of airborne radon are also affected by patterns of opening windows and the presence and frequency of use of whole house, bathroom or kitchen fans, stove hoods, wood stoves, oil or gas burners and clothes dryers.

RADON PATHWAYS INTO STRUCTURES

Radon may enter structures as a gas from uranium-containing rock or soil, dissolved in well water emerging from taps and showers, or to a lesser extent, in building products made from such rock.

Airborne Radon

It is a principle of physics that a gas will move from an area of higher concentration or pressure to one of lower concentration or pressure. Thus radon in the cracks of rocks in an underground mine will move into the air of the mine cavity and, if a passageway exists, radon in the soil or rocks beneath a structure will move into the structure.

Radon gas may enter a building through cracks or gaps in a basement or foundation, through dirt floors and holes from form ties and sumps (Fig. 3-4) (49). Soil is the major source of radon in indoor air. This is supported by the fact that the parts of buildings closest to the ground have the highest levels. For example, in 85 homes tested in a University of Maine study basements had twice the radon concentration of other living areas and 8 times the concentration of ambient air (50).

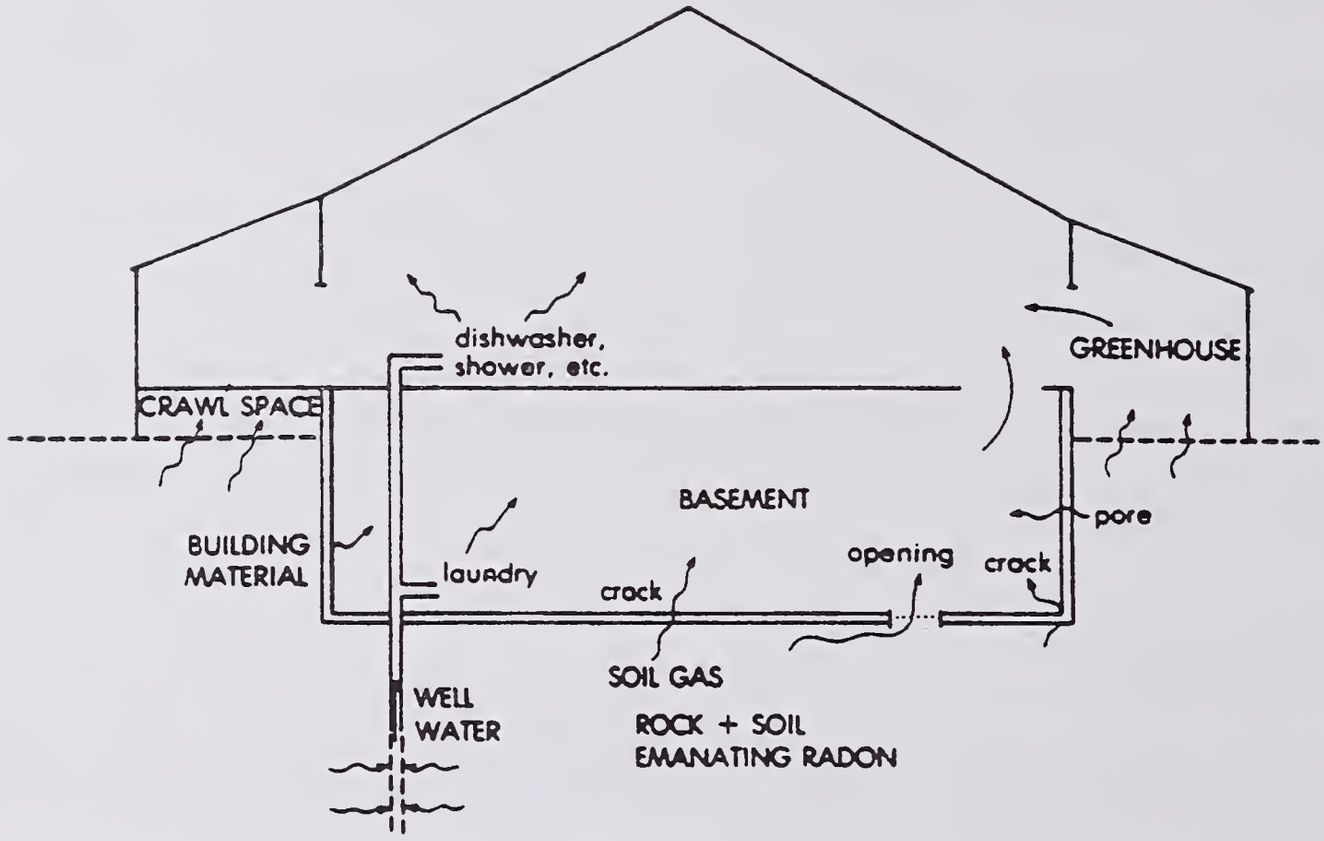


Figure 3-4. Pathways of Radon into Structures

(Source: University of Maine and Maine Department of Public Health, 1983)

Radon in Water

Surface water concentrations of radon are low and are further reduced by exposure to air since radon simply moves into the air. In contrast, well water which has passed through uranium bearing rock can carry radon into a

building. This radon can be ingested when people drink or prepare food with such water; however, inhalation of radon and its entry into the lungs is of greater concern. As water emerges from taps and showers radon is released into the air of a kitchen, bathroom or laundry. Agitation increases the rate at which radon is emitted.

Other Sources

Radon is emitted by concrete or brick made from uranium bearing rock or sand (8, 9). Building materials are not considered a major source of radon (49) but may contribute significant amounts in certain cases. Solar heated structures with rock used for heat storage or large stone fireplaces or walls are potential sources of radon, because more soil and rock is used in their construction. Small amounts of radon can also enter structures in coal, oil or natural gas and be emitted from poorly vented cooking or heating systems.

RADON IN STRUCTURES: SCOPE OF THE PROBLEM

Indoor radon has been reported in Finland (51) and Canada. In the United States elevated levels have been found in every state. While the average readings in a state or region were often low, in many cases a significant fraction of the buildings had readings above the EPA action level, or an annual average reading of 4 pCi/1. Since the country has not yet been thoroughly surveyed using statistically valid techniques it is unknown how many areas with high levels exist. Of almost 10,000 homes in Canada, 264 (3%) were above 4 pCi/1 and 49 (0.4%) were above 10 pCi/1 (25).

Federal and Nationwide Studies

A study by Lawrence Berkely Laboratories for the DOA included a compilation of field data. Analyis from 20 studies covering a total of 1377 homes from 38 regions of the country where there was no previous expectation of high radon levels indicated an average reading of 1.5 pCi/1, and 1-3% of the homes had levels of 8 pCi/1 or more. Homes with the highest levels were located in eastern Pennsylvania, Fargo, North Dakota, the Spokane River valley of eastern Washington and northern Idaho as well as parts of Colorado, Idaho, Montana, Tennessee, Maryland, Florida and Maine. Few used statistically based sampling procedures but most selection processes had a strong random component, although about 1/3 were done because of prior information suggesting a probability of high radon concentrations (55, 63).

Terradex Corporation of California measured radon levels in the usual living areas of homes which are not associated with man-made sources of radium or uranium contamination. Alpha track detectors were used. Over 30,000 homes were tested. The data is grouped by 5 regions labeled northeast, midwest,

northwest, southeast and mountain states. Measurements were made from 1980 through December 1986, and each varied from a week to a year. In some cases more than 1 detector was placed in a house and time of year for the measurements varied (25).

Data was accumulated from Terradex clients and therefore is not random. Larger numbers of readings are from known hot spots in New Jersey, Maine and Pennsylvania. Most of the very high measurements came from the northeast region which includes the above 3 states, along with Massachusetts, Vermont, New Hampshire, Connecticut, Rhode Island, New York, Delaware, Maryland, and the District of Columbia (25).

The northeast has the highest average, 11.1 pCi/1, considerably higher than the mountain states with 6.5 pCi/1 and more than twice the midwest average of 5.1 pCi/1. Notably, both the northeast and mountain states contain granitic mountain ranges. Forty-four percent of the homes in the Northeast had readings greater than the 4pCi/1 EPA action level, and 25% had readings greater than 8 pCi/1. Pennsylvania had the highest percentage of homes with readings above 4 pCi/1 (56%) (25).

When New England states are considered separately, Rhode Island has the highest average reading at 12.4 pCi/1 and the greatest number of homes with readings above 4 pCi/1 (30%). The Rhode Island average, however, is based on relatively few readings - only twenty. Massachusetts has the lowest average at 2.4 pCi/1, with 17% of homes above 4 pCi/1 (25).

EPA initiated systematic collection of data on radon levels in single family residences during the winter of 1986-87. A cooperative program was developed between EPA and 10 states (64). Over 20% of the 11,600 homes tested contained radon levels above EPA's action level. The percentage of homes above 4 pCi/1 was highest in Colorado (39%). The other states and their associated percentages were: Wisconsin (27%); Wyoming (26%); Kansas (21%); Rhode Island (19%); Connecticut (19%); Kentucky (17%); Tennessee (16%); Michigan (9%); and Alabama (6%). It is notable that the 2 states with the lowest percentages of homes with readings over 4 pCi/1 had the 2 highest single readings - Alabama with 180 pCi/1 and Michigan with 162 pCi/1. This suggests that areas with low average amounts of radon might still have hot spots (5, 64). The data suggests all 10 states may have a significant fraction of homes with radon levels of concern (64). The worst hot spot identified is Clinton, New Jersey, a town of 2000, where 105 homes sited above a uranium-laden limestone cliff had readings over 4 pCi/1. Thirty-five of these measured over 200 pCi/1 and 5 were above 1000 pCi/1.

Statewide and Regional Studi

The New Jersey Department of Environmental Protection study showed a statewide average of 5.4 pCi/1, with measurements ranging from 0.1 to 246 pCi/1. Thirty-three percent of homes had levels greater than the EPA action level implying need for further testing or remedial action. The state averages are elevated by the high readings along the Reading Prong. One

hundred and sixty-five public buildings were also tested including schools, hospitals and municipal buildings around the state. Radon levels were found to be generally lower than in homes, but 13% were above 4 pCi/1 (65).

In the Ohio Regional Air Pollution Control Agency study 163 locations were sampled beginning in early 1986. The average radon concentration was 7.0 pCi/1, with 21% of homes above 10 pCi/1 (60).

The BPA monitored radon levels in over 250 homes in Washington, Oregon, Idaho, and Montana for several months. First floor living areas averaged 1.20 pCi/1 with levels ranging from 0.05 to 15.0 pCi/1. Four percent of these had levels above 5 pCi/1 (5).

New England Studies

Of 85 homes tested by the University of Maine, 30% exceeded EPA's action level. Elevated radon levels in water were found in some homes (50).

The joint survey of radon levels in indoor air and well water by the Connecticut Departments of Health Services and Environmental Protection assessed the predictive value of hydrologic and household parameters, and the relationship of radon levels in the well water to levels in indoor air. EPA's method of water sampling was used in 262 homes and alpha track devices were placed in the living areas of 202 of these.

Radon levels in indoor air ranged from 0.1 to 24.6 pCi/1, with a mean of 2.0 pCi/1. 11% had levels of 4.0 pCi/1 or greater. Well water ranged from 100 to 130,240 pCi/1. Twenty-six percent of wells had levels of 10,000 pCi/1 or above. Regression analysis indicated 17% of airborne radon was attributable to water.

RADON IN MASSACHUSETTS

Uranium Content of Rock and Soil

As indicated previously EPA studies of the bedrock geology of Massachusetts suggest a potential for high indoor radon levels in some areas. Uranium content of rocks and soils may vary in different parts of the Commonwealth due to different bedrock types, for example, granite rocks tend to be higher in uranium content than the sedimentary rocks found in Boston. Even rock types with generally low uranium levels may have localized pockets or "hot spots" (59).

Indoor Radon

Several thousand buildings, mostly homes, have been tested for radon in Massachusetts though most of the buildings were not randomly selected. Massachusetts homes tested in the Terradex study averaged 2.4 pCi/1; however, 17% were above the EPA action level.

The University of Pittsburgh measured radon in homes in Middlesex and Essex counties in Massachusetts. The average readings in the main living areas were similar to the 1.4-1.5 pCi/1 found in other studies, and were lower than around the Reading Prong and some southern and western states. However, 15% of the houses had readings of 3 pCi/1 and over, and 1% were 15 pCi/1 or more (61). Some very high readings were found including the earth-encased concrete house in Beverly.

Boston's Channel 7 (WNEV), a CBS affiliate, sponsored a project in which homeowners throughout Massachusetts were invited to participate in a radon testing program. Fifty-three (30%) of the 174 readings were above 4 pCi/1; 9 readings (5%) were above 20 pCi/1 (66).

The DPH Radiation Control Program (RCP) has a database of approximately 3000 sample results. Again 14 to 25% of the readings are above EPA's action level of 4 pCi/1 and 1% are above 20 pCi/1. Thus far no hot spots have appeared (67).

While none of the above may have been random samples it is notable that 10-30% of the readings were above the action level (assuming the University of Pittsburgh data to be 10%). Taking 20% for purposes of argument, this could suggest 320,000 homes in the state might have elevated radon levels. Since the samples were non-scientific and only 3000-4000 readings are on record a large degree of uncertainty remains about the scope of elevated indoor radon in Massachusettes.

Radon in Water

There appears to be little problem with radon in public water supplies in Massachusetts. In 1981-1982 EPA and DPH studied groundwater fed public water supplies in 100 towns, covering 90% of Massachusetts residents who drink public groundwater. The water was virtually free of radon. Many towns using groundwater store it in standpipes or reservoirs where the radon dissipates (61). The Quabbin Reservoir has never shown a radon problem (68).

Risk Characterization

Estimates of the risk of dying from lung cancer as a result of radon exposure are based on models which assume a dose over a period of time and

take into account the fact that people only spend a portion of their time in the structure in question. For example, an EPA model assumes 75% of the time is spent at home for a lifetime of 70 years. As Table 3-2 shows, at 4-20 pCi/l 1-5 out of every 100 people would be expected to die from lung cancer. At 20-200 pCi/l, 6-21 deaths would occur and at 200 pCi/l 44-77 deaths would result. In the latter case, if the same 100 persons lived only 10 years in a house with 200 pCi/l, only 14-42 deaths would occur (49). All of these are very high risk factors. Governmental action is usually considered appropriate when the risk is 1 in 100,000. It should be noted that the EPA action level is somewhat arbitrary. Public health concern should not stop at 4 pCi/l.

About 4 out of every 100 people die of lung cancer. The Surgeon General estimates about 85% of lung cancer deaths are due to smoking (49) and EPA's Science Advisory Board estimates that about 10% are due to radon (49). Radon could cause roughly 100,000 deaths per year. Radon in water may be responsible for about 1000 deaths each year (9). The risk of a smoker dying of lung cancer is increased considerably if the smoker is also exposed to elevated levels of radon (18).

It is useful to compare the risk of lung cancer death from radon exposure to that of smoking (Table 3-2). For example, persons exposed to 4 pCi/l have 5 times the risk of a non-smoker; 200 pCi/l is 60 times the non-smoker risk and equivalent to that of a 4 pack a day smoker (49). These estimates could be low. An NRC study released early in 1988 (18) suggests the lung cancer risk from lifelong exposure to radon is several times greater than earlier estimates.

RADON MITIGATION

The radon problem appears to be resolvable. Unacceptably high levels of indoor radon can generally be lowered in existing structures and prevented in new ones. Homes in Pennsylvania and New Jersey reading 3000-4000 pCi/l have been improved to an annual average below the EPA action level of 4 pCi/l (5). The goal of mitigation is to reduce indoor radon to at least 4 pCi/l. Mitigation and prevention techniques, however, may vary in complexity, cost and effectiveness. In addition, mitigation may be required on an ongoing basis since structures age, settle and new cracks appear.

Indoor airborne radon levels are determined by the differences between radon's rate of entry and its rate of removal. The rates of infiltration of soil gas and escape of radon from tap water can be reduced by source control methods which prevent, deflect or reduce radon's entry. An increased removal rate is achieved through increased ventilation (5).

Source Control

As indicated earlier, air which is naturally or mechanically driven from a building in an effort to lower radon concentrations lowers the inside air pressure. Pressure is also lowered by clothes dryers, the chimney effect of warm air rising through a building and out openings at the top, wind blowing around the house, and fireplaces and furnaces. When air pressure is lowered, radon, which is at concentrations in the soil thousands of times greater than in the air, will be drawn into structures (70).

A number of treatments have been proposed or used to lower the rate at which radon enters a living space. There is little scientific information available regarding the effectiveness of some of these techniques although theoretically they would seem to be effective (50) and may require special procedures.

Venting crawl spaces should dilute radon before it enters living areas (5). Sealing untrapped drains or floors over crawl spaces, caulking cracks or openings in slab floors, and placing airtight barriers between floors and crawl spaces are straightforward techniques. A floor drain or sump pit can be vented outdoors using a dryer hose and small electric fan (51).

A system used in some houses with full basements is to sink pipes through the basement floor and connect them to an exhaust fan. This both draws radon laden air outside and offsets the house's tendency to draw air from the basement to rooms above by decreasing the pressure beneath the basement (51). Using this technique, EPA has turned the homes on the New Jersey cliff with the 10 highest readings into a research lab for mitigation methods. Of these 6 are now reading below 4 pCi/1 (71).

Most public water supplies come from reservoirs where radon escapes into the air, whereas private supplies usually come from groundwater and are more likely to contain radon. Activated carbon filters, depending on type, can remove up to 99% of waterborne radon and also remove potentially toxic uranium salts. The unit is functionally similar to a water softener and contains a backflushing system to remove accumulated material from the filter. Recent research, however, has suggested that backflushing can release radon progeny and there is concern developing about disposal of the unit.

Aeration systems are available which spray water inside a large container. Air is blown through it and the radon is vented outdoors. This can remove up to 99% of the radon but is costly.

Removal

Radon concentrations vary with the number of air changes per hour which are lower in winter when structures are kept closed. The average annual ventilation rate in single family houses has been reported at 0.5-1.5 ach

(53). New homes vary from 0.2 to 0.5 ach due to their tighter construction (9). Energy efficient buildings may be as low as 0.1 to 0.2 ach (69).

Doubling the ventilation rate in relation to a fixed source rate decreases radon concentration by 50% according to the dilution principle (Fig. 2-3). Ventilation methods used to reduce radon levels are the same as those used in the reduction of indoor air pollution in general: passive ventilation and active ventilation in the form of fans (both exhaust and intake) and air-to- air heat exchangers (50).

Tightly-sealed houses may have high radon levels in spite of a relatively low rate of soil gas infiltration. The ventilation rate can be increased significantly if several windows are kept open a little in winter. Using ventilating fans or opening windows in bathrooms or other water use areas is also effective when water is a significant source (50). Tightly sealed houses with balanced ventilation are optimal.

Removal of airborne particles via a fan connected to a filter, decreases the total number of particles in a room. This leaves a larger proportion of radon decay products unattached. However, there is a potential for increased lung doses of radiation from these increased numbers of unattached progeny. This might occur with any techniques employing selective removal of aerosols through filtration thus the net effect of filtration is unclear (53).

One quarter of the buildings constructed on phosphate tailings or mineral land and one-half of those constructed on uranium tailings require special construction methods (9). Controls such as special construction or alterations have a cost. Additional natural or mechanical ventilation uses more energy. Fans and heat exchangers involve capital and maintenance costs. High efficiency air cleaners for recirculated air reduce levels of particulates and associated radioactivity, but are costly. Surface coatings for basement walls and floors which are being tested could be considerably less costly than other measures. A Colorado Department of Health study of effectiveness of control measures found dilution by ventilation to be the most cost effective alternative (53). Adding a vented crawl space has been suggested as the most cost effective way to minimize radon infiltration in new buildings (53).

RADON: FEDERAL INITIATIVES

EPA is regarded as the lead agency for indoor air quality. Statutes under which it might regulate radon include: TSCA (aimed at toxic pollutants); UMTRCA (residential projects on tailings); SDWA (for water-borne radon); CERCLA (for radioactive substances on hazardous waste sites); and very importantly, SARA (which in 1986 gave EPA explicit authority to conduct research and disseminate information on radon and other indoor air pollutants).

The Indoor Radon Abatement Act (Public Law 100-551), amending the Toxic Substances Control Act (TSCA), was signed into law October 1988. The Radon

Act requires EPA to establish a radon information clearinghouse, to study radon levels in schools and federal buildings, and to develop construction standards. The Act's 45 million dollar, 3 year budget also provides financial and technical assistance for state radon programs (72).

Nationwide Database

Both EPA's uranium bearing rock and soil mapping program and DOE's bismuth monitoring program are aiding predictions of where geological factors might lead to high indoor radon levels.

EPA's joint study with the states continued during the winter of 1987-88. Homes were tested in 7 more states including Massachusetts. In addition to the 10 states involved in the 1986-87 EPA study, 11 are independently surveying for radon - Alaska, Florida, Idaho, Ohio, Illinois, Indiana, New Jersey, New Mexico, New York, North Carolina, and Virginia. The Indian Health Service is testing reservations in Michigan, Minnesota and Wisconsin (26). According to deputy EPA Administrator A. James Barnes, these results and those of epidemiological studies will combine to give EPA "a robust database in another couple of years" (64).

A separate and somewhat duplicative national survey, to be conducted by EPA, was mandated by the 1986 amendments to the Superfund Law. Its purpose is to determine the distribution of indoor radon levels throughout the U.S. in homes, schools and workplaces. Detectors were projected to be in place in 2000-5000 homes across the country by late 1987 (64).

Health Effects and Risk Assessment

In terms of number of potential cancer deaths a year, radon is EPA's greatest environmental problem: more severe than hazardous waste and toxic chemicals (5). EPA estimates 4 to 8 million homes in the United States exceed the action level (64).

A number of epidemiological studies are underway to investigate the link between radon in homes and lung cancer. The National Cancer Insititute (NCI) is undertaking projects in New Jersey and Missouri; Argonne National Laboratory has a study in progress in Pennsylvania (64). The multi-year Argonne study is examining lung cancer in women living in eastern Pennsylvania, where particularly high indoor radon levels occur (26).

EPA is developing an Integrated Risk Information System (IRIS) which is a computer-based chemical-specific risk assessment and management program. The National Air Toxics Infomation Clearinghouse has a complementary data base. IRIS presumes some knowledge of health sciences but not technical expertise (73).

The Office of Health and Environmental Research (OHER) has sponsored radon research for several decades including animal studies, and radon transport and diffusion into and within structures. It has also supplemented previous Public Health Service uranium miner studies with research on New Mexico miners which includes more accurate exposure data (1).

Other Efforts

EPA's Radon/Radon Progeny Measurement Proficiency (RMP) Program assists states and the public in selecting companies with competence in measuring radon levels. Detector operations and data management quality are evaluated semi-annually. The program's objective is to promote standard measurement and quality assurance procedures (74). RMP approves laboratories as well as businesses (49).

One hundred radon-proof homes are being built in varying geological situations along the Reading Prong of New Jersey. This is a joint effort of EPA, the National Association of Homebuilders Research Foundation, the New Jersey Builders Association and the State Department of Community Affairs which regulates the housing industry (44).

The National Conference of State Legislatures has received an EPA grant to provide information and assistance to states developing radon programs. The project is entitled State Radon Programs: The Role of Legislation. The grant will supply funding for research and a report on the nature and scope of the problem, outlining existing federal and state programs and presenting policy options (75). EPA's regional offices offer radon information and assistance and an information hotline is envisioned (9).

The Federal Emergency Management Agency (FEMA) may allow disasters attributable to radon to qualify for Federal aid under the Disaster Relief Act.

The CIAQ radon working group co-chaired by EPA and DOE, will coordinate DOE and other programs. The DOE and EPA programs are coordinated with other countries through the Commission of the European Communities (1). CIAQ and EPA have co-sponsored international conferences on indoor air quality in Berlin, Stockholm and Amherst.

Standards, Regulations and Guidelines

EPA has reponsibility for standards regarding airborne ionizing radiation and has developed guidelines for homes built on uranium mine tailings (40CFR192). These guidelines suggest a maximum radon level of 4 pCi/1 (or 0.02 WL of radon decay products) (9).

There are currently no federal regulations or guidelines for indoor radon levels for structures built on natural soil (9). EPA recommends trying to reduce radon levels to annual average exposure of 4 pCi/1 (0.02 WL) or lower

(49) and taking action if levels are above 4 pCi/1 or exposure approaches 1 WLM/year (51). EPA suggests that the higher the radon level, the greater the urgency to confirm the reading and take action (Table 3-1) (9).

EPA also proposes that certain factors be considered in determining tolerable indoor levels, such as the presence of smokers or children, the percent of time spent at home, whether someone sleeps in the basement, and how long the family expects to live in the home. Indoor levels of radon progeny may increase since they attach to particulates from tobacco smoke. Children have also shown greater sensitivity to other radiation types. EPA recommendations include: discouraging smoking, spending more time in areas with lower radon concentrations, opening windows, using fans, and keeping crawl space vents open (49).

Exposure guidelines have been criticized as arbitrary and misleading because there is evidence that the level of radon progeny in the air can double if any of the inhabitants smoke. In the absence of particulates, ions adhere to walls and furniture where they pose no health threat. But if ions adhere to smoke particles they are slowed down by a factor of 10 and may be inhaled (76). The EPA recommended maximum level was set in part because EPA feels it is difficult to reduce levels much below 4 pCi/1 (64).

A range of guidelines and standards has been proposed for indoor radon by others (5). Unlike statutes and regulations, guidelines do not carry the force of law. Standards may either be advisory or carry regulatory force if referenced in legislation. In 1984 the National Council on Radiation Protection and Measurements (NCRP) recommended a standard of 0.055 WL in existing buildings and suggested remedial action at greater than 2 WLM per year (8 pCi/1) (77). The World Health Organization has established 0.11 WL for existing structures (9). ASHRAE recommends 0.01 WL in new buildings. ASHRAE has also proposed ventilation standards which have been adopted as a minimum in Sweden, Denmark and France. No standards have been set for water-borne radon (55).

RADON: STATE, MASSACHUSETTS AND LOCAL INITIATIVES

A number of states have taken steps to better define the extent of elevated radon levels. In most cases action has been the result of legislation requiring a statewide survey.

A few states have gone further. New Jersey began a $4.2 million comprehensive initiative in 1986. New York and Pennsylvania are spending similar amounts. New Jersey, which estimates 1.9 million homes may have unsafe levels, has the most comprehensive state program to date, calling for a state radon survey and an epidemiological survey to determine if radon causes cancer. Homes of 1200 living cancer patients will be tested. The program will also include confirmation of test results through free second opinions, monitoring, certification of home improvement and testing firms, and an information and outreach program (26).

The number of firms in New Jersey offering radon testing increased from 3 in 1985 to 120 in 1987. There were none doing mitigation in 1985, 22 in 1987. The state oversees and regulates testing firms. New Jersey has strict licensing and certification standards. About half the applicants for certification have been rejected. The state offers free second opinions of test results. New Jersey and Pennsylvania have toll-free radon hotlines staffed by scientists. Both have produced pamphlets and brochures. New Jersey is doing periodic public opinion surveys. In northern New Jersey 80% of real estate transfers involved a radon test in mid-1987.

New Jersey plans to implement regulations requiring the licensing of radon testing and radon mitigating companies. Although New Jersey officials acknowledge that there have been few complaints of fraud, they fear the absence of regulations could encourage fraudulent or unreliable firms.

Pennsylvania and New Jersey are also providing low interest loans to assist home owners with modifications to reduce radon. The Pennsylvania program which has been in existence for 2 years, offers 3 million dollars or up to $7,000 per home with graduated interest rates based on income and free radon tests to homeowners on the Reading Prong (5, 78).

Radon Testing

There is currently no structured radon testing program in Massachusetts and all sampling is based on consumer requests. Those contacting RCP are provided with a list of testing firms (61).

While DPH requests that homeowners report test results, such reporting is entirely voluntary. Some contractors are providing results to the RCP; however, location is identified only by zip code. RCP has built a database of around 3000 reported sample results.

Reports of elevated radon levels (> 4 pCi/1) receive a graded series of responses, adapted from EPA's recommendations (Table 3-3). Readings are confirmed with charcoal measurements. An alpha track detector is then installed for several months to a year. When results above 20 pCi/1 are obtained, grab samples may be taken and water tested in homes with private wells. Above 4 pCi/1 remedial action is recommended.

RCP has reprinted a radon booklet published jointly by the EPA and the Center for Disease Control and is also distributing 20,000 booklets and fact sheets and giving presentations to local Boards of Health, the media, realtors and others (49).

EPA's Eastern Environmental Radiation Facility has been used to analyze air and water samples. However, as demand increases, federal facilities are becoming less available to states. The State Laboratory Institute's facilities might be used but they need considerable additional equipment and supplies, including charcoal canisters and scintillation vials.

Table 3-3 DPH Response to Indoor Radon Levels

Radon Concentration (pCi/l)	Radon Progeny Concentration WL	Response
>4-20	>0.02-0.1	reading confirmed with charcoal measurements; alpha track detector then installed for 1 year.
21-100	>0.1-0.5	site visit to take charcoal and grab sample readings; alpha track detector installed for 3 months; if private well, water tested.
>100	>0.5	as above but alpha track detector may be left in place for a shorter period; remedial action recommended.21-200

(Source: Massachusetts Department of Publis Health. "A Citizen's Guide to Radon: What it is and what to do about it," 1986)

EPA produces an annual Radon Measurement Proficiency Report which lists home radon testing services which meet EPA's accuracy criterion of 25%. A recent consumer group study of seven EPA-approved testing programs found that three of the seven did not meet this 25% accuracy standard in at least one of three rounds of testing. Because the radon canisters had been exposed to the equivalent of 20 pCi/1/liter, their lab analysis should have been straightforward, according to a representative from Buyers Up, Ralph Nader's Public Citizen coalition which conducted this survey (79).

EPA forewarns radon companies prior to evaluating their accuracy. To insure high, consistent standards in the future, EPA will institute secret, blind testing of these companies in March 1989.

Joint EPA-DPH Radon Survey Study

DPH regards radon as a serious health problem and, in conjunction with EPA, participated in a seven (7)-state survey of radon levels in residences. The other states involved in the study were Arizona, Indiana, Minnesota, Missouri, North Dakota, Pennsylvania as well as Indian lands in Missouri, Minnesota and Wisconsin.

In September 1988, EPA and the Surgeon General issued a joint advisory, calling for radon testing in most U.S. homes. This advisory was issued in conjunction with the results of the seven-state survey, which demonstrated that nearly one third (1/3) of all homes tested had screening levels of radon in excess of EPA's 4pCi/1 action level. Combining the results of the seven-state survey with a prior EPA ten-state survey, over three million homes in the seventeen (17) states tested had screening levels of radon greater than 4 pCi/1.

In Massachusetts alone, 1,659 radomly selected homes were tested. Of these homes, one in four had radon screening levels exceeding 4pCi/1. See Figure 3-5 for the radon survey in Massachusetts. The highest levels were recorded in Middlesex, Essex and Worcester counties.

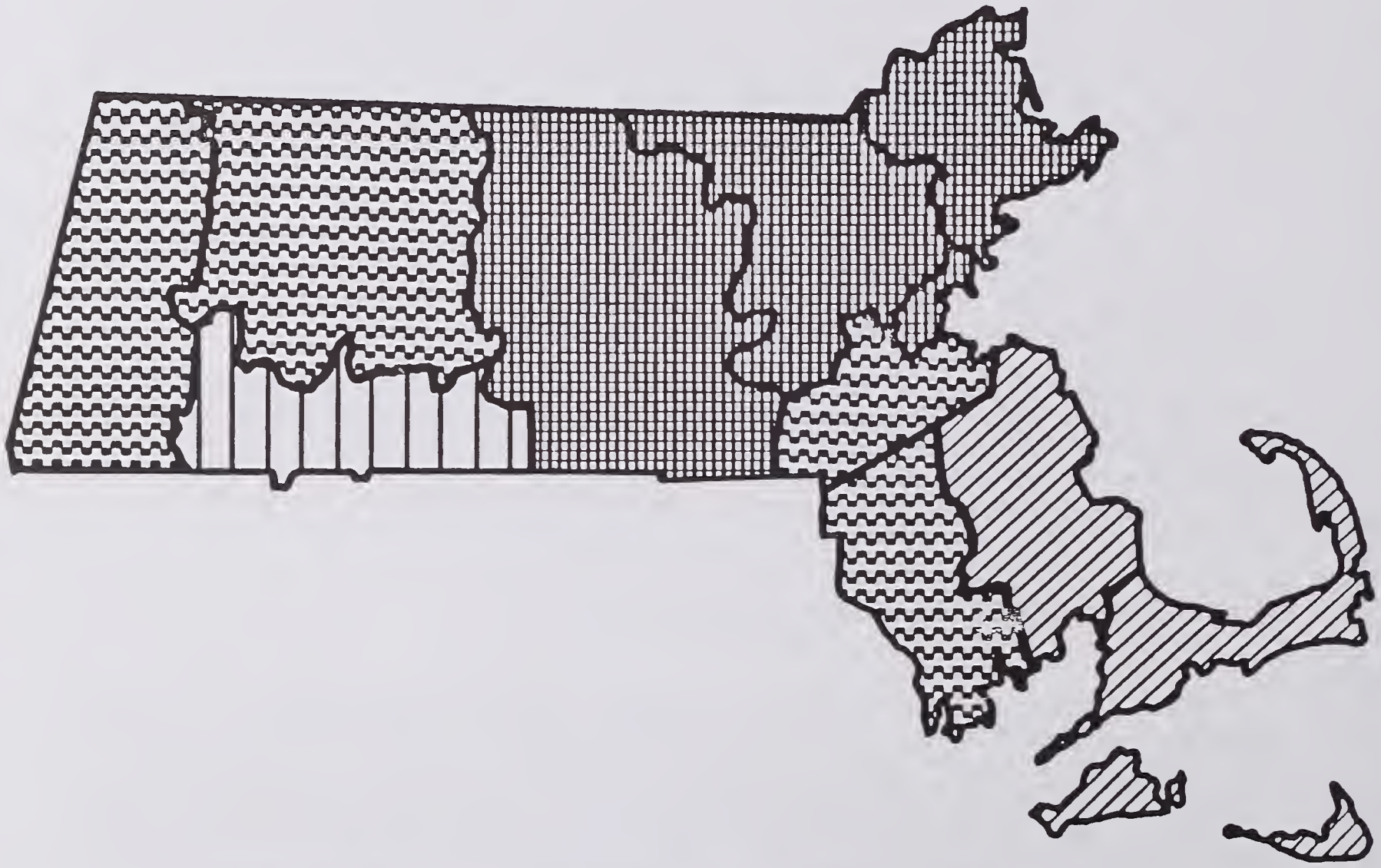
The distribution of radon screening measurements in Massachusetts is shown in Table 3-4. For those homes with screening levels of 4 to 20 pCi/1, EPA recommends retesting and if a level in excess of 4pCi/1 is confirmed, appropriate mitigation should be implemented. For those homes with screening level readings in excess of 20 pCi/1, immediate mitigation is recommended.

Massachusetts: Standards, Regulations and Guidelines

There is currently no research underway regarding health effects of radon exposure and there are no state radon regulations or water testing standards. Local boards of health (BOH) regulate radiation and have the authority to promulgate regulations, but are awaiting state guidelines. BOH may also make a determination of whether a house is habitable, depending upon whether radon levels pose a public health risk. DPH has received requests from BOH for assistance in radon study design and is providing presentations and literature.

FIGURE 3-5

Massachusetts Radon Results by Region



Estimated Percent of Houses With Screening Levels Greater Than or Equal to 4 pCi/L

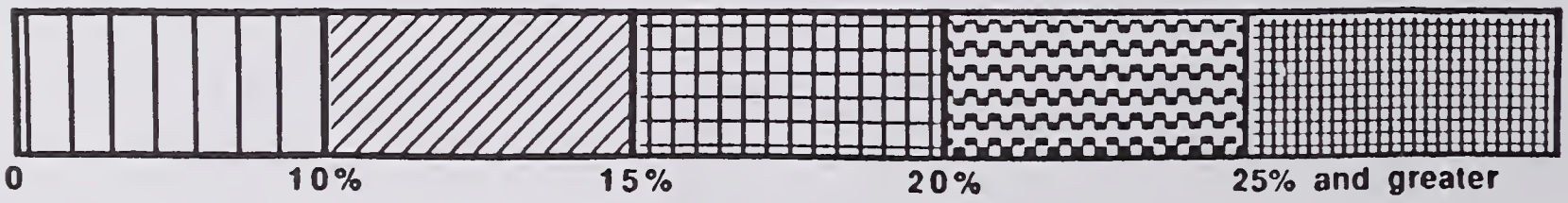


TABLE 3-4

Massachusetts

Distribution of Indoor Radon Screening Measurements

Radon Levels, pCi/L	Percent of Houses with These Levels*
< 4	76
4 - 20	23
> 20	1

Average Level	3.4
Number of Houses Measured	1659

* There Is A 95% Certainty That These Values Represent Homes In Massachusetts Within 3 Percentage Points

Chapter 4: FORMALDEHYDE

FORMALDEHYDE AND ITS SOURCES

The Compound Formaldehyde

Formaldehyde is the simplest member of the chemical group called aldehydes, and consists of one oxygen and two hydrogen atoms attached to a carbon (Figure 4-1) (80). Like all aldehydes formaldehyde occurs as a vapor at room temperature (80). Formaldehyde is a colorless gas with a characteristic pungent odor. Although some sensitive individuals can detect the odor of formaldehyde at approximately 0.05 ppm, most people only detect concentrations of 1.0 ppm or greater (81). Formaldehyde is highly reactive and dissolves in water, methanol and other substances (44).

Formaldehyde occurs naturally as an intermediate product in animal metabolism and is produced in the decay of animal and plant matter (82) and the combustion of fuels such as wood and oil.

Ambient air may contain formaldehyde from biological decay, vehicle exhaust and industrial emissions (82). Indoor air additionally may contain formaldehyde from varius biological and industrial uses, numerous manufactured products, and as a result of combustion of fuel or cigarettes. Formaldehyde has been used as a preservative and disinfectant in hospitals and biological laboratores for 100 years (83). As a preservative it is often used as formalin, a mixture of 35-50% formaldehyde in water (44, 80).

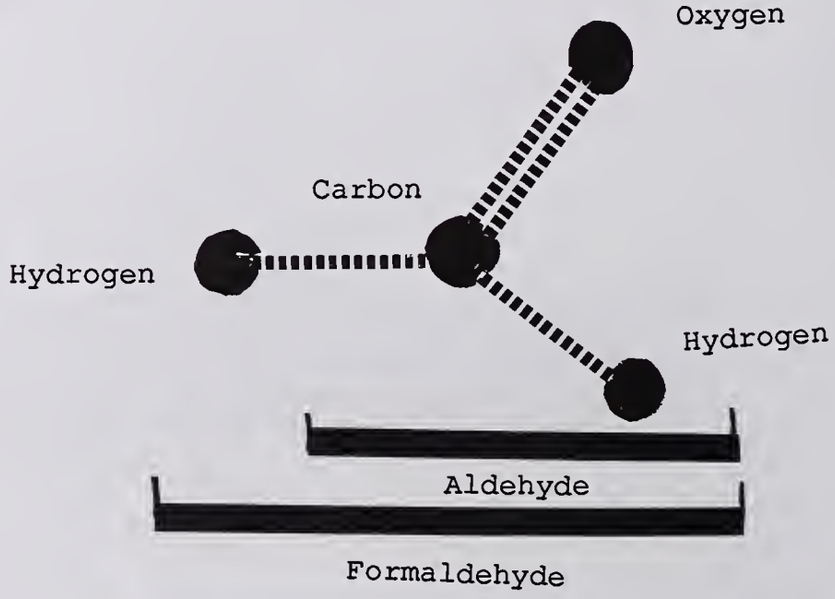


Figure 4-1

The Structure of Formaldehyde

Most formaldehyde is produced inexpensively by laboratory methods. Formaldehyde is used or occurs in many consumer products either alone (free) or as part of other compounds. Half the formaldehyde produced today is used in the production of urea formaldehyde (UF) and phenol formaldehyde (PF) resins which are in turn used as bonding and laminating agents in wood products, in wall insulation as UFFI, and for modifying the properties of textiles or paper (Table 4-1) (44). Each of these products emits formaldehyde, at a rate dependent upon the nature and properties of the source. For example, emissions from UFFI decrease over time while emissions from cigarettes are constant.

Bonded Wood Products

Synthetic resins containing formaldehyde are used mainly as adhesives in bonded wood building materials including paneling, plywood and particleboard, wallboard, hardboard partitions, and ceiling panels (44). UF resin, a prepolymer made from a mixture of urea, formaldehyde and water, is the most common adhesive (84). Heating (curing) causes numerous different formaldehyde emitting compounds to form (85). All bonded wood products with UF resin release formaldehyde.

Release of unreacted formaldehyde from UF bonded wood products has been known since Fahrni invented particleboard in 1943. However, exposure risk was at first low because the products were not widely used. Over the next several decades concern increased over the loss of forests and a dwindling wood supply. Bonded wood products found a larger market because they allowed more efficient use of harvested trees. Formaldehyde release problems arose when use of particleboard and hardwood plywood paneling increased (83).

In 1973 teachers refused to work in a new classroom building in Karlsrhue Germany becaues of an irritating odor. The problem was found to be large amounts of particleboard plus the fact that the ventilation system had been turned off for several weeks. Similar problems have been reported in other countries (83). Particleboard was introduced about 10 years later in North America than in Europe since America had larger forest reserves (85).

Over the past 20 years bonded wood products have been used more and more instead of whole wood. There are three major types: particleboard, medium density fiberboard (MDF) and hardwood plywood. Particleboard is composition board with 6-10% resin by weight, along with small wood particles and additives. Over 90% of the resin is UF. Seventy percent of particleboard is used in cabinets, furniture, fixtures and other furnishings, with the remainder for construction in mobile as well as conventional homes (85).

MDF contains only UF resin which is 8-14% of its weight. Ninety-five percent of MDF is used for doors, furniture, cabinets and fixtures. MDF and particleboard differ in that MDF particles are separated via cooking or shredding into fibers less than 1 mm long. MDF is fine, appears more like wood than particleboard, and may be worked to achieve smooth edges (85).

Table 4-1 Common Sources of Formaldehyde

- Acrylic
- Air and furnace filters
- Air fresheners
- Aircraft parts
- Antihistamines
- Antiperspirants
- Antistatic agents
- Ashtrays
- Automotive brakes
- Barber and beauty shop disinfectants
- Binders
- Biocides
- Blanket controls, bases and covers
- Brake drums
- Cigarette smoke
- Coated papers used for cartons and labels
- Coatings
- Coatings for appliances
- Compactors
- Cookware handles and knobs
- Counter and table tops
- Dental bibs
- Dental filling
- Deodorants
- Detergents
- Diaper liners
- Dinnerware
- Disinfectants
- Door panels
- Drapery and upholstery fabrics
- Drinking milk
- Dyes
- Electrical connectors
- Electronic equipment
- Embalming agents
- Examining table paper rolls
- Facial tissues and napkins
- Fan blades
- Faucets
- Fiberboard
- Flour preservative
- Formica
- Glues
- Hair-setting solutions
- Hair-waving preparations
- Hand pumps
- Hardboard
- Hardware
- Heat sensor switches
- Hospital bed sheets
- Insecticides
- Kidney dialysis procedure
- Knobs and buttons
- Lawn and garden equipment
- Maraschino cherries
- Mascara and other cosmetics
- Nail hardener
- Nail polish
- Oil-based paints
- Orthopedic casts and bandages
- Orthopedic procedures
- Paint and wood finishes
- Particleboard
- Permanent-press cotton
- Pesticides
- Pharmaceuticals
- Plastic
- Plumbing fixtures
- Plywood
- Portable tools
- Preservatives
- Pressed wood furniture
- Pressure gauge bodies
- Primer coat for automobiles
- Radiators
- Radio and TV bases and housing
- Rayon
- Roofing
- Sewing machine parts
- Shampoo
- Soap dispensers
- Softwood plywood
- Sporting goods
- Stove and refrigerator hardware
- Textile waterproofing
- Tire rubber
- Toilet seats
- Toothpaste
- TV/radio/stereo cabinets, door panels, store displays, kitchen cabinets
- Utensil handles
- Vaccines
- Vacuum cleaner parts
- Water-softening chemicals
- Wax and butcher wet strength paper
- Wheat grains and agriculture seeds
- Wool

Hardwood plywood is laminated with formaldehyde resin which bonds the layers of wood and veneer. It is 2.5% resin by weight. About 55% of this plywood is used for paneling, 30% for furniture and cabinets and the rest for doors and flooring. Furniture often contains particleboard on the inside and

veneer on the outside. Softwood plywood also contains formaldehyde (44, 85).

Urea Formaldehyde Foam Insulation

UFFI is another important source of formaldehyde emission. Problems with formaldehyde in indoor air are commonly associated with UFFI. UFFI became increasingly popular during the early 1970s and was in wide use by the end of the decade (44).

Formaldehyde, especially UFFI, has led to thousands of complaints to the CPSC (81). UFFI is produced on site by the installer. UF based resin, compressed gas (usually air), a foaming agent, and an acidic hardening agent are combined to make a foam with the consistency of shaving cream. UFFI is pumped into a wall cavity through a hose. UFFI is 75% water by weight at the time it is inserted and can take up to several months to cure and become solid, depending upon temperature, ability of the cavity to vent the water, the chemicals used and other factors. When set, UFFI has the appearance of an open-celled foam. It usually shrinks and cracks when dry, allowing air to circulate, and may eventually crumble. UFFI has generally been used to retrofit older homes (82, 86).

Other Sources

Formaldehyde resins are widely used in the textile industry for clothing, linens, draperies and upholstery fabrics. UF resin is used for color fixation since it increases the adherance of pigments to cloth. The crease resistent (permanent press) characteristics of nearly all linen and cotton come from formaldehyde as do shrink and flame resistance, and water proofing (44).

Wallpapers, especially those which are prepasted or made with fibers or layers of paper are bonded with formaldehyde resins. UF resin is used to impart wet strength to different grades of paper. It is found in paper products such as facial tissues, napkins, paper towels, waxed paper, and grocery bags. The Food and Drug Administration (FDA) has allowed UF treated paper to contact food since 1972 (44).

Formaldehyde is used in personal care items including cosmetics, shampoos, and disposable sanitary products. It is also found in room deoderizers, starch-based glue, fiberglass insulation, ceiling tile and floor coverings (44, 85).

HEALTH EFFECTS OF FORMALDEHYDE EXPOSURE

Formaldehyde is a public health issue because exposure produces numerous physiological effects and because it is virtually impossible to escape in

modern living environments. While the potential for exposure to high levels of formaldehyde (several ppm) is less in the home than in industries or occupations which produce or use formaldehyde, residential exposures are important because more time is spent there and a greater number of people are exposed. Formaldehyde in building materials, furnishings, textiles and paper products increases exposure (44) as does the presence of tobacco smoke.

Public concern about formaldehyde exposure initially focused on its irritant properties, and more recently on the effects of long term exposure, particularly formaldehyde's potential as a carcinogen (44).

Physiological Action

Formaldehyde is a normal metabolite in humans and other animals. It is a vital ingredient in the synthesis of essential biochemical substances and not toxic in small quantities (12). Measureable amounts of free formaldehyde are seldom found in plasma or tissue (44).

External formaldehyde can enter the body through inhalation, ingestion, or adsorption by the skin. Ninety-five percent of the formaldehyde inhaled through the nose is absorbed in the upper respiratory tract. At low concentrations in "clean" air most of the formaldehyde is absorbed in the front third of the nose. Formaldehyde which is inhaled into the lungs may, after a series of reactions, be oxidized to carbon dioxide and exhaled or be excreted with urine. Some is incorporated into tissues. Because it is water soluble formaldehyde can easily enter the bloodstream. Formaldehyde displays similar behavior in all mammalian species (44, 81).

Range of Effects

Formaldehyde has been established as an irritant in both humans and other animals. Residential and occupational exposures have reportedly resulted in prolonged eye, nose and throat irritation, wheezing, coughing, diarrhea, nausea, vomiting, headaches, dizziness, lethargy, irritability, disturbed sleep, and olfactory fatigue. Skin contact can lead to acute inflamation, irritation, contact dermatitis and hives. Exposure can lead to bronchial asthma (40) and menstrual irregularities (Table 4-2) (44, 81). High concentrations can be fatal.

It is hard to relate specific health effects to specific formaldehyde concentrations because the responses and complaints of individuals vary. Response thresholds vary by whether a person is hypersensitive, healthy or diseased. Complaints of consumers cannot always be ascribed to formaldehyde.

Tolerance to formaldehyde odor may develop after several hours of exposure, but repeated exposure may lead to hypersensitivity (12). In addition, each effect occurs at a wide range of concentrations. As the

concentration of formaldehyde increases, both the severity of reaction and the number of people responding increases.

Table 4-2 Formaldehyde Concentrations and Adverse Effects: Occupational and Residential Studies

ppm	Effect	Type of exposure
0.0—10	Nausea, eye, nose and throat irritation, headache, vomiting, stomach cramp	Residential
0.02—4.15	Diarrhea, eye and upper-respiratory tract irritation, headache, nausea, vomiting	Residential
0.09—5.6	Burning of eye and nose, sneezing, coughing, and headaches; 3 of 7 suffered from asthma or sinus problems	Occupational
0.3—2.7 Av. 0.68 Median 0.4	Annoying odor, constant pricking of mucous membranes, disturbed sleep, thirst, heavy tearing	Occupational
0.13—0.45	Burning and stinging of eyes, nose, and throat, headaches	Occupational
0.2—0.45 Av. 0.36	Irritation of eyes and upper respiratory tract, drowsiness, headaches, and menstrual irregularities	Occupational
0.13, 0.57, and 0.44	Headaches, concentration problems, dizziness, nausea, coughing, increases in recurring infections of the upper respiratory tract, and irritation of eyes, nose, and throat	Schools
~0.83	Loss of olfactory sense, increased upper respiratory disease, subatrophic and hypertrophic alterations in nose and throat, ciliostasis of nasal mucosa, increased absorptive function of nasal mucosa	Occupational (greater than 5 years to less than 10 years)
0.9—1.6	Itching eyes, dry and sore throats, disturbed sleep, unusual thirst upon awakening in the morning	Occupational
0.9—2.7	Tearing of eyes, irritation of nose and throat	Occupational
?	Chronic airway obstruction, respiratory tract and eye irritation, small decrease in pulmonary function during workday and work week	Occupational
1.3—3.8	Menstrual disorders, pregnancy complications, low birth weight of offspring	Occupational

(Source: Gammage, R.B. et al. Indoor Air Quality, CRC Press, 1984)

Information regarding the effects of formaldehyde on humans comes from controlled exposure studies. Low level exposure from 10 minutes to 5 hours under controlled conditions leads to eye, nose and throat irritation at 0.2 ppm and above (44). These effects generally disappear whenever exposure stops (5). At low concentrations the major effect of formaldehyde is irritation of the eyes and mucous membranes. Effects usually begin to appear between 0.05 and 1.5 ppm. Here non-specific complaints such as thirst, dizziness, headache, tiredness and difficult sleeping are reported.

EPA has determined irritation of eyes (burning and tearing), nose, throat and lungs occurs for the majority of people at 0.1 to 1.1 ppm (87). Eye irritation was reported at 0.05 ppm and above. Beginning at 1 ppm nose, throat and bronchial irritation occurred.

Headache, nausea, coughing, chest constriction, rapid heartbeat and pressure in the head occur at 1-2 ppm (5) as well as drowsiness, vomiting and diarrhea. Concentrations greater than 5 ppm are likely to produce wheezing, coughing and chest constriction (81). Formaldehyde levels of 50 ppm have produced bronchial inflammation, pulmonary edema, pneumonia and death (12). Table 4-3 shows the concentrations of formaldehyde at which effects have occurred in controlled human and animal studies. (44).

Table 4-3 Lowest Effective Concentration: Controlled Studies

ppm	Length of exposure	Species	Effect
0.01	5 min	Human	Eye irritation
0.05—0.06	min	Human	Odor threshold
0.07	min	Human	Optical chronaxy threshold
0.08	1.5 months	Rabbit	Changes in evoked potential of optic nerve
0.08	min	Human	Threshold to affect the functional state of cerebral cortex
0.2	1 hr	Human	Eye, nose, and throat irritation
0.25	5 hr	Human	Dryness of nose and throat, decrease in mucous flow rate
0.31	1 hr	Guinea pig	Increased airway flow resistance, decreased compliance
0.55	10 min	Rat	Reduction in respiratory rate
0.83	3 mon	Rat	Histological and histochemical changes in cerebral amygdaloid complex
0.83	1 min	Human	Altered functional state of cerebral cortex
0.83	90 days	Rat	Peribronchial and perivascular hyperemia, lymphohistiocytic proliferation in lung, focal hyperplasia and RE system activation in cerebral cortex
0.83	10 min	Human	Irritation of upper tract and eyes, accelerated breathing, EEG changes, such as alpha rhythm enhancement, changes on automatic nervous system
0.83	10 months over 2 generations	Rat	Morphological changes in upper respiratory tract, decreased liver weight
0.83	Continuous beginning 10—15 days before mating	Rat	Increase in size and number of extramedullary hematopoietic centers, increased epithelial proliferation of common bile duct, increased abnormalities of renal epithelium
1.4	1 min	Human	Eye sensitivity to light lowered in unacclimated group
1.67	Continuous or intermittent	Guinea pig, rat	Sensitization (inhalation) leukocytosis and change in blood cholinesterase
2	6 hr/day, 5 day week for 18 months	Rat	Epithelial hyperplasia, squamous cell metaplasia of nasal turbinates, rhinitis
3.8	90 days continuous	Rat, dog, rabbit, monkey, guinea pig	Death in 1/15 rats; some inflammation in lungs in all species
4.1	4 hr/day on days 1—19 of gestation	Rat	Increase in threshold of neuromuscular excitability, peripheral white blood cells, decreased hemoglobin, and rectal temperature in pregnant animals
4.2	1 min	Human	Unbearable without respiratory protection
5.6 and 14.3	6 hr/day/week for 24 months	Rat, mice	Carcinogenic and other pathological changes of the nose (see text)
15.5	10 hr	Mouse, rabbit, guinea pig	5/7 mice, 3/5 rabbits, 8/20 guinea pigs dead: closed eyes, slow deep respiration, convulsions
41.5	1 hr/day, 3 day/week for 35 weeks	Mouse	Upper respiratory tract inflammation, basal cell hyperplasia, epithelial stratification, bronchopneumonia
50	1 hr/day, 1 day/wk for 18 months	Hamster	Squamous cell metaplasia
482	4 hr	Rat	L. C. 50 (approx.)
0.2	Min in water	Human	Elicited allergic dermatitis reponse in a sensitized subject
3.7—37 g/ℓ and 3.7 g/ℓ as challenge solution	Intermittent patch induction	Human	Induced dermal sensitization in 4.5 to 7.8% of the healthy test subjects

(Source: Gammage, R.B. et al. Indoor Air Quality, CRC Press, 1984)

The NAS Committee on Toxicology concluded there is no population threshold for irritant effects of formaldehyde. NAS suggests most healthy adults will not experience acute toxic effects at formaldehyde concentrations less than 0.1 ppm and less than 20 percent will experience reactions at concentrations less than 0.25 ppm. However, individuals vary in their susceptibility. Infants, people with respiratory problms, allergic individuals and the elderly may respond at lower levels or experience more severe effects (86).

Most sensitive individuals can detect the odor of formaldehyde at approximately 0.05 ppm. A few may even experience allergic reactions at these levels. About 50% of healthy people detect formaldehyde at 0.17 ppm - a concentration which may also irritate the eyes and upper respiratory tract (83). Most people can detect formaldehyde at a level of 1.0 ppm (5).

Sensitive Individuals and Sensitization

High sensitivity to low levels of formaldehyde occurs in 10-20 % of the population. Some susceptible people experience asthmatic symptoms at low concentrations (5). About 4% of the population is particularly sensitive to developing allergic dermititis when skin is exposed (83).

In addition to its action as an irritant in both humans and animals, formaldehyde also has sensitizing effects (44). Repeated exposure to liquid or vapor formaldehyde can sensitize certain individuals. When re-exposed they may have an allergic skin reaction which can include oozing and other effects, or mild or severe asthmatic reaction. Responses may increase in intensity over time. The NAS Committee on Aldehydes has indicated persons sensitized to formaldehyde and with hyperactive airways (10-12% of the U.S. population) have more severe responses (44).

Formaldehyde as a Human Carcinogen

A link between formaldehyde and cancer has not yet been definitively shown in humans. However, the Federal Panel on Formaldehyde and several federal agencies have suggested formaldehyde be considered a human carcinogen until proven otherwise (5). This decision was based on evidence that formaldehyde causes cancer in animals and limited evidence that it causes cancer in humans. Nasal cancers in animals and limited data from human studies have led EPA to classify formaldehyde as a Group B1 probable human carcinogen.

Stewart et al. analyzed the cause of death of over 4300 workers who were employed at 10 industrial facilities which produced or used formaldehyde between 1938 and 1965. They compared exposed workers to non-exposed workers at the same facilities and to the general population in the same counties. Cause of death did not correlate with length or level of formaldehyde exposure, although lung cancer incidence was higher than expected (201 cases

compared to 182 expected). Excessive nasal cancers were not found nor were excesses of the same cancers found by anatomists, pathologists and embalmers: brain cancer and leukemia (87).

The above study was jointly conducted by NCI and the Formaldehyde Institute. When the data was reanalyzed by recalculating exposure levels, job categories and employee groupings, and controlling for certain factors, a clear increase in risk of lung cancer was found at average exposure levels greater than 0.5 ppm. In addition, a suggestive (though statistically insignificant) increase in cancers of the mouth and throat was found for workers in plants which produce formaldehyde resins (89).

Acute Toxicity Studies in Animals

Formaldehyde exposure has been studied in mice, rats, guinea pigs, rabbits, dogs, cats and monkeys. Formaldehyde tends to act the same way in all mammalian species.

The effects of formaldehyde exposure on rats, cats and mice range from slightly toxic if applied orally to moderatly toxic if inhaled. Formaldehyde causes mild to moderate irritation of the skin in rabbits. Liquid formaldehyde is a severe eye irritant causing corneal injury and edema in rabbits while vapors cause tearing and discharge but no injury. Half of a group of guinea pigs became sensitized when formaldehyde was reapplied to their skin 2 weeks after an initial application (12).

Extended Studies in Animals

Animals exposed to formaldehyde for 3 to 35 weeks developed pathological tissue changes. In rats and guinea pigs repeated skin contact or inhalation led to sensitization. Exposure to high doses caused salivation, vomiting, cramps and death of test animals (44).

In one 90 day continuous inhalation study, rats, guinea pigs, rabbits, monkeys, and dogs exposed to 3 ppm all developed various degrees of lung inflammation (12). In another study groups of 25 rats were exposed continuously to formaldehyde for 45-90 days. Animals exposed for 90 days to 8.1 ppm developed respiratory and eye irritation, decreased food intake and decreased liver weight (12).

In a 13 week study by the Chemical Industry Institute of Toxicology (CIIT) 20 mice and 20 rats were exposed to 4, 12.7, 38.9 ppm of formaldehyde for 6 hours a day, 5 days a week for 13 weeks. The 4 ppm group showed no adverse effects. At 12.7 ppm, decrease in weight and nasal erosion occurred in 2 rats. At 38.6 ppm damages to the nasal membranes led to termination of the experiment after 2 weeks.

Pathological Tissue Changes

Pathological tissue changes in animals caused by formaldehyde include changes in the nasal membranes, mutations, teratogenic and carcinogenic effects. Studies of dogs and rats to determine teratogenic effects showed no gross malformation, slight increase in the percentage of stillborn puppies, slight decrease in weight gain, and some changes in lymphoid and liver tissue. Formaldehyde leads to mutations in bacteria, fungi, insects, and mouse cells (but not in whole mice). It has caused chromosome breakage and recombination in yeast, insects, some cultured cells of mammals, and in rats (44).

In another CIIT project, mice exposed for 1 hour a day, 3 times a week for 35 weeks to 41 ppm of formaldehyde then exposed to 123 ppm for 29 weeks, developed non-tumerous cell proliferation. A Formaldehyde Institute study of continuous inhalation showed a similar effect in rats and monkeys (12).

Formaldehyde exposure has led to cancer in mice and rats. Early studies of the cancer inducing potential of formaldehyde in rats, mice and hamsters gave negative results but the studies were questionable because of the doses used, duration of exposure, and other factors (44).

In one study, Fischer rats were exposed to formaldehyde vapor at 0, 2, 6, and 15 ppm for 6 hours a day, 5 days a week for 18 months. Thirty-six of the 120 rats developed cancer in the nasal cavities at 15 ppm. This was the first study to show formaldehyde to be a potential carcinogen. CIIT reported in 1980 that nasal cancer had been found in rats exposed to 6 ppm for 24 months and mice at 15 ppm for 24 months (44).

A recent New York University study of rats supports the hypothesis that formaldehyde causes cancer in animals. Rats were exposed to 14.6 ppm of formaldehyde. After 24 months 45 % developed nasal cancer, while none of the controls did. This was the same type of nasal cancer as found in the CIIT and a previous New York University study (44).

Formaldehyde may be a carcinogen when in combination with other compounds. When it was administered to rats along with benzo(a)pyrene and diethylnitrosamine, it did not increase the incidence of tumors with the former but did with the latter (44).

FORMALDEHYDE MEASUREMENT AND MONITORING

Three quantities important to measure with respect to formaldehyde are emission rate, concentration in the air and the total exposure to which an individual is exposed. Because emission rates and air concentrations change and are a function of many variables, personal monitoring at breathing level is the best method of determining total exposure. Personal monitoring is

tedious and expensive, but is performed occasionally in industrial situations (83).

Emissions Testing

Four general types of emission measurement procedures are used for products which release formaldehyde such as bonded wood products and UFFI. These are static, dynamic chamber, equilibrium chamber and distillation/ extraction tests. Each is described briefly below.

Static tests rely on diffusion of formaldehyde gas from the sample to a medium in which it is collected. The dessicator test is commonly used and uses a tight glass container, a rack for samples, and a beaker or plate to hold the collection medium which is a measured quantity of distilled water (Fig. 4-2) (85). This system is maintained at 75 degrees Farenheit for 2 to 24 hours with longer time periods used for materials with lower emission rates. The amount of formaldehyde absorbed by the water is converted to an emission rate using a calibration table.

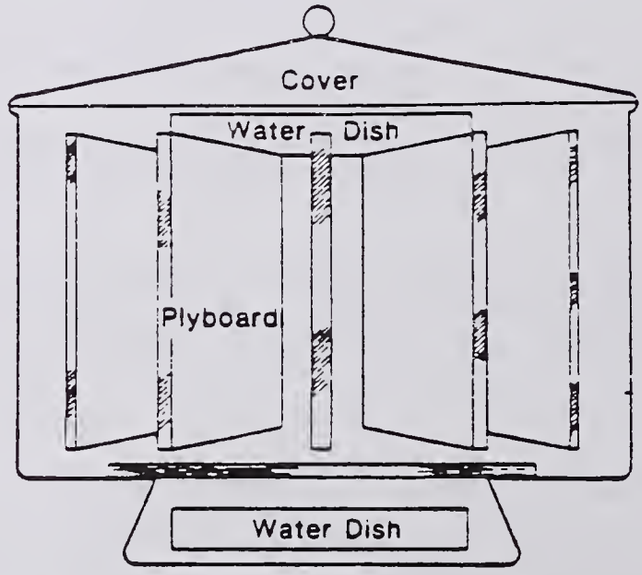


Figure 4-2.

Desiccator Test Apparatus

(Source: "EPA Final Report: Formaldehyde Exposure in Residential Settings: Sources, Levels, and Effectiveness of Control Options," 1986)

The amount of formaldehyde emitted by a sample and absorbed by water can also be determined using analytic methods such as the chromotropic acid, purpald, acetylacetone and pararosaniline procedures.

Dynamic chamber tests pass air at a controlled rate past a sample in a chamber. Formaldehyde is carried out of the chamber with the air. This technique models air contamination in a room. Chambers may be laboratory size or large enough to hold a full size sample of a material such as particleboard.

Equilibrium chamber tests are similar to static chamber tests. The test material is placed in a closed container without any air change. After an equilibrium is reached between the formaldehyde concentration in the material and the air, the formaldehyde content of the air is measured.

The distillation/extraction test most often used involves using a boiling solvent such as toluene to collect the formaldehyde. Samples are placed in the boiling toluene. Then the toluene with formaldehyde is bubbled through distilled water to extract the formaldehyde.

The formaldehyde surface emission monitor does not destroy the sampled matter and is portable (Fig. 4-3). It consists of a covered fine brass sieve which is placed about 2.3 cm above the test substance. A solid sorbent material is sprinkled on the sieve, and the system is sealed for two hours. The sorbent is then washed with distilled water from which the formaldehyde is collected. This monitor is sensitive enough to detect emissions from any substance known to affect indoor formaldehyde concentrations.

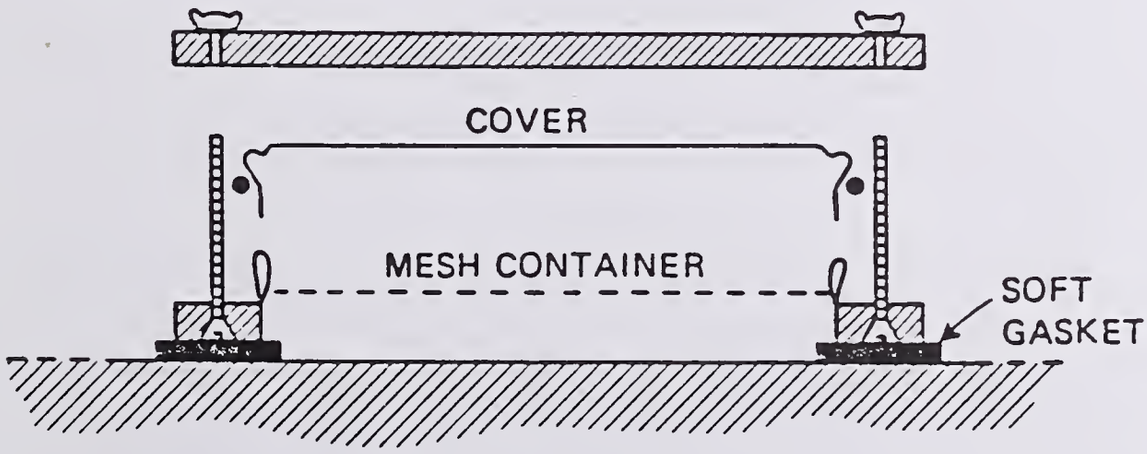


Figure 4-3

Formaldehyde Surface

Emission Monitor

Monitoring Air Concentrations and Personal Exposure

Concentrations of airborne formaldehyde can be obtained from formaldehyde surface emission monitors by determining the emission rates, the areas of various emitters, the volume of living space, and the measured air exchange rate (44).

Air concentrations are measured using the NIOSH standard method 3500 (adapted from P&CAM 125), also referred to as the "chromotropic acid" method. The working range of this method is 0.02 to 0.4 ppm for a 90 liter air sample. It is the most sensitive formaldehyde method in the NIOSH Manual of Analytical Methods, against which all other sampling methods are validated. Using this method, sampling would take 90 minutes at an air flow rate of 1 liter/minute. In addition, a passive dosimeter has been validated against the NIOSH method 3500 to +/- 25% when exposed for 168 hours (7 days), with a lower level of detection of 0.02 ppm. DPH uses both of these techniques.

DEQE's Air Quality Surveillance Branch recommends indoor air monitoring for formaldehyde and related aldehydes with impinging air sampling techniques using duplicate sampling trains. One technique samples room air; the other functions as a reference sample with charcoal filtered air and distilled water. The air pumped through this sample train is mixed with specific chemical reagents and produces a characteristic color, which can be measured against a standard.

The Air Quality Surveillance Branch also uses a portable toxic gas monitor wet-chemistry analyzer to perform this task. This monitor can be set-up for indoor as well as ambient air real-time monitoring. The analytical scheme is as follows: air is sampled through a calibrated air pump built into the instrument at a rate of 0.5 liters/minute. Any aldehydes (formaldehyde) present will react with two chemical reagents, sodium sulfite and pararosaniline, to produce a colored product and measured in a photocell, yielding real-time part per billion concentrations. (Linear range of instrument is 0-250 ppb).

Information as to the presence of formaldehyde in room or ambient air can be obtained through the use of Draeger colorimetric tubes. These tubes detect pollutants by manually pumping air through the chemically reactive resin, producing a net color-change proportional to the concentration in parts per million.

Another method for sampling for aldehydes is found in the EPA Compendium of Air Toxics Methods, section TO-5. This method involves ambient air sampling using impinger samplers containing absorptive solutions, which are then extracted using solvents to isolate the formaldehyde. Formaldehyde levels are determined with high performance liquid chromatography.

Several air sampling devises are available which also may be adapted as personal sampling devises to determine total individual exposure. Most widely used is a dry sulfite device. Its sensitivity limitations require 5-7 days of

exposure. A passive bubbler monitor passes formaldehyde through a permeable membrane where it is absorbed by 3-methyl-benzothiazolone hydrazone. The manufacturer indicates it can detect 0.1 ppm through a 2 hour exposure. Another which is in the development stage, contains a film of hydrobenzoic acid hydrazide which forms crystals when exposed to formaldehyde. These can be "read" visually. Pumped tube methods, which are adequately sensitive, use solid sorbents. One of these the Tejada method, was developed by EPA and is being used in its Toxics Air Monitoring System (90).

FACTORS AFFECTING FORMALDEHYDE LEVELS

The level of formaldehyde in a room depends on the rate at which it is being emitted, and the rate at which it is being removed. Emission rate relies on many factors while removal is a function of ventilation or air cleaning factors. As discussed below, however, there is an interactive effect between existing formaldehyde concentration, ventilation rate and emission rate.

Formaldehyde Emission

The rate at which formaldehyde is emitted by a source depends upon the nature, age and load factor of the source, temperature, humidity, and the concentration of formaldehyde already present.

Nature of Source

Materials vary in formaldehyde emission rate (5). This is due to differences in the formaldehyde compounds they contain, certain physical characteristics and their condition. Products with PF rather than UF resins emit considerably less formaldehyde. Fibrous glass insulation and ceiling tile with PF resins are unlikely to increase indoor levels by more than 0.02 ppm. (85). Products which have been treated to reduce emissions have lower rates (44).

Plastics which are molded, high density, extruded or non-porous release little formaldehyde (44) whereas UFFI, which is porous, emits high levels. Drapery and upholstery fabric treated with UF resins have low emission rates of only about 0.015 mg/sq m/hr which could raise indoor levels by more than 0.01 ppm in very high loading situations.

Combustion of both petroleum and plant products releases formaldehyde. Thus formaldehyde is a product of burning fuel for cooking or heat and smoking tobacco products. For example, gas ovens and ranges can emit from less than 2 to nearly 30 mg/hr (85). In one test an oven and stove top burner emitted 25 and 15 mg/hr respectively (44). Gas space heater emissions range from less than five to over 60 mg/hr depending on burning efficiency and new kerosene space heaters emit up to six mg/hr. Combustion appliance data shows

formaldehyde emission rate depends on whether the appliance is properly tuned and functioning. Data for sidestream cigarette smoke show formaldehyde emissions ranging from 0.2 mg to nearly 1.5 mg per cigarette (85).

Age of Source

The rate of formaldehyde emission decreases as materials cure and age. Most likely half the formaldehyde in most materials is released within 2-5 years. Bonded wood products contain and emit both free formaldehyde and formaldehyde bonded into resins. Free formaldehyde is the residual formaldehyde left after the paneling or particleboard is hot pressed for curing. Free formaldehyde can be released rapidly and most formaldehyde initially released is of this type. Formaldehyde release is proportional to the total residual formaldehyde in a product and emission decreases exponentially with age. Generally formaldehyde release due to compounds breaking down occurs in lower amounts over a longer period. The various forms of bound formaldehyde differ in their rates of decomposition and susceptibility to reacting with water (44, 83, 85). Therefore they will differ in emission rate.

Similarly, emission rates of UFFI generally decline with time. Most UFFI installations release the greatest amount of formaldehyde in the first year (85). The Connecticut Department of Health Services surveyed formaldehyde levels in 30 UFFI homes in 1986. These were selected from 500 homes previously showing high levels. Ten controls without UFFI were used. Most homes insulated with UFFI prior to 1981 had levels at or near those of the controls. A few still had levels up to 0.3 ppm. Age of insulation was inversely correlated with formaldehyde levels (89). New apparel has very high emission rates, up to 0.03 mg/sq m/hr. Laundering decreases the emission rate (85).

Load Factor

Load factor is the ratio of a product's surface area to indoor air volume. The greater the surface area per unit of air volume the higher the emission rate (83). Adding new furnishings such as cabinets or furniture containing bonded wood products can increase total emissions of formaldehyde by increasing the load factor. Mobile homes often contain higher formaldehyde levels than conventional homes because more formaldehyde-containing particleboard and plywood are used in their construction (5). In UFFI homes the insulation has a very high surface area and therefore high emissions.

Temperature and Humidity

An increase in temperature, humidity or both leads to an increase in formaldehyde emission. The emission rate increases exponentially as temperature rises (Fig. 4-4). This response occurs relatively rapidly. In fact, emission rate has been found to track daily solar temperature variations (Fig. 4-5) (83).

As amount of humidity in the air increases, the amount of formaldehyde emitted increases accordingly. However because of the high moisture capacity

of wood and the slow moisture exchange rate between air and wood, moisture equilibrium may take a long time to achieve (83). The emission response to increased humidity is thus much slower than the termperature response and is believed to be linear (85).

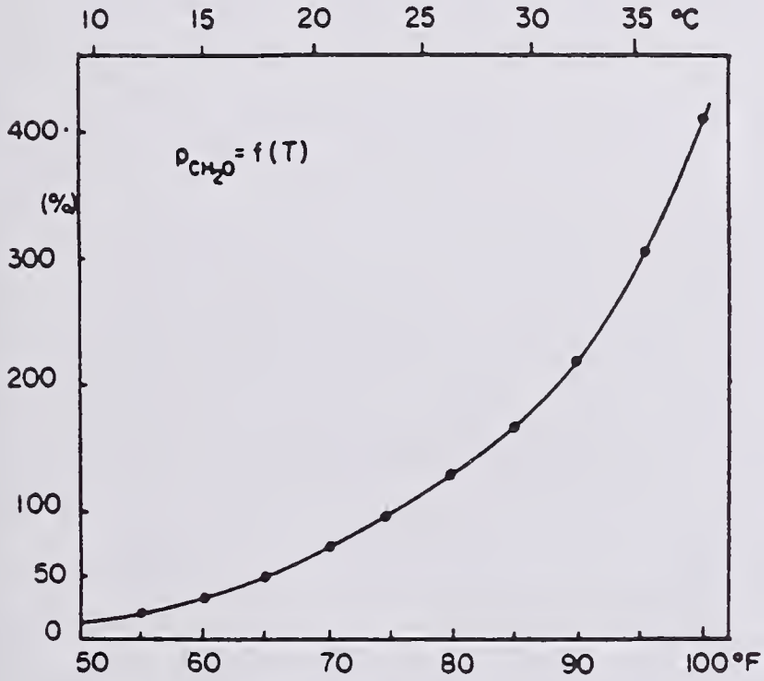


Figure 4-4. Formaldehyde Emission from Pressed Wood Products as a Function of Temperature

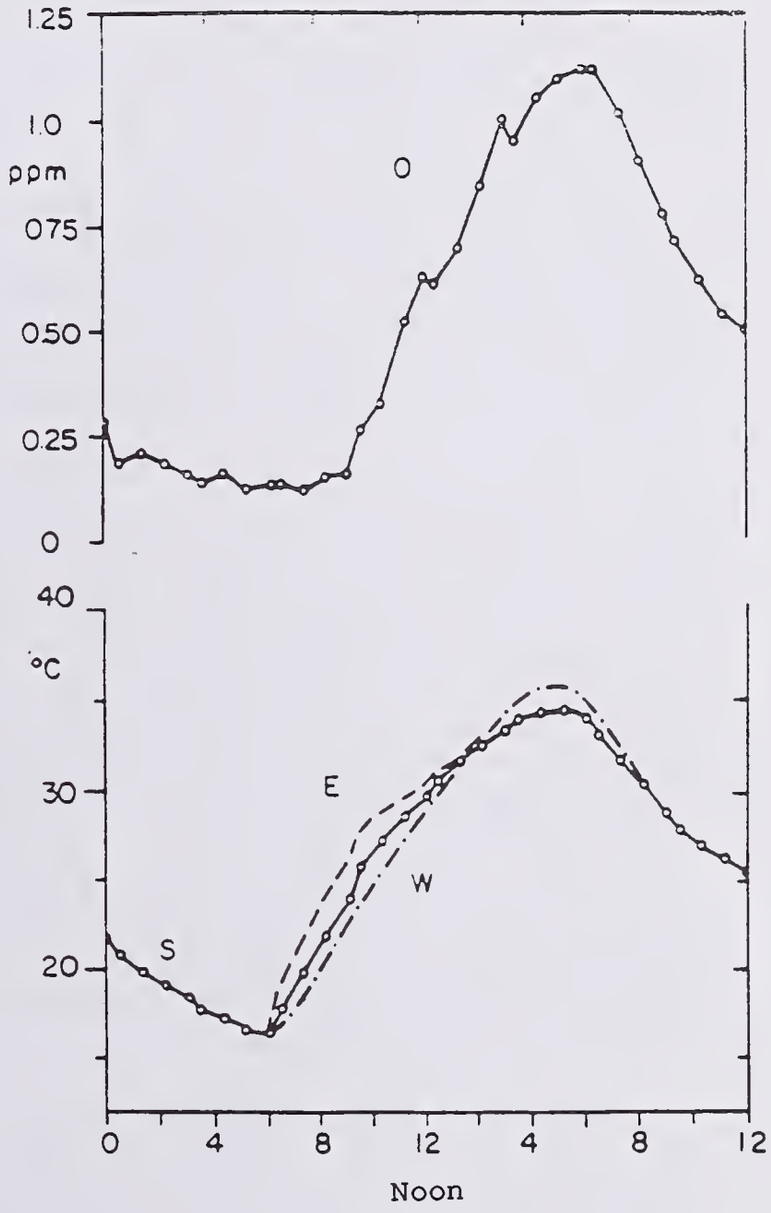


Figure 4-5.

Daily Variation of Formaldehyde Concentration and Wall Temperature in a Mobile Home. E = east wall, S = south wall, and W = west wall.

(Source: Meyer, B. and Hermanns, K. Journal of the Air Pollution Control Association, 1985)

Formaldehyde readily combines with water to form methylene glycol and is attracted to areas where moisture is present such as air conditioning ducts, cold outside walls and areas where wood is present (wood contains water). Moisture itself enhances the release of formaldehyde from resins (83).

Concentration

The emission rate of a product is inversely related to the formaldehyde concentration in the surrounding air. Formaldehyde, like any gas, will move from areas of higher to lower concentration (44). Formaldehyde will be emitted into the air of a formaldehyde-free test chamber or closed room at an ever decreasing rate until equilibrium is reached. Thus, emission rate is both a component of concentration and affected by concentration.

Ventilation and Air Cleaning

Ventilation has a direct effect on both formaldehyde concentration and emission. Test chamber studies have been used to study the relationship among these factors (44). As the number of ach increases and formaldehyde levels drop, emission rate increases (Fig. 4-6). The relationship however is non-linear (83, 85). Doubling the ventilation rate may reduce airborne formaldehyde concentration by 33-38% as opposed to the 50% increase expected. Air cleaning techniques which lower formaldehyde concentration presumably have the same effect.

Interactive Effects

A study in Oak Ridge, Tennessee examined the effects of emission factors and ventilation on formaldehyde levels. Twenty four-hour integrated monitoring was used to assess the factors determining formaldehyde levels in 40 houses representing a diversity of ages, construction types, heating and insulation types (including UFFI) (44).

Indoor formaldehyde levels fluctuated with time. Daily variations occurred as a result of occupants' activities and the solar cycle. Studies of a 3 year old prefit UFFI house indicated formaldehyde levels increased in the middle of the day as the sun heated the walls. By contrast in a ten year old non-UFFI house (Fig. 4-7), maximum concentrations occurred in the early morning when the house was closed and people were asleep (44).

Levels of UFFI varied with the seasons in one UFFI home. In winter drying and cooling of the UFFI (Fig. 4-8) decreased formaldehyde concentrations after they had increased when the heat was first turned on. Levels increased in the spring but opening windows then led to a decrease (44).

formaldehyde levels may be 0.14 - 1.36 ppm in the parts of the lab where the students are working compared to 0.03 - 0.36 ppm in other areas of the room.

Anatomy labs where human cadavers are used range from 0.4 - 6.0 ppm. Other parts of schools and colleges generally have formaldehyde levels of less than 0.05 ppm (44).

Building Contents or Structure

By 1983 3 billion square feet of particleboard was being produced each year in the United States, as well as 2 billion square feet of hardwood plywood and 600 million square feet of MDF (85). Around 300,000 metric tons of UF resin a year are used for these pressed wood products. Most buildings now contain from 0.2 to 1.0 square meter of product per cubic meter of air. UF bonded products are now the most abundant formaldehyde emitting products in North America. Melamine formaldehyde bonded plywood is not used much and PF bonded exterior grade plywood emits little formaldehyde (83).

In the Tennessee study formaldehyde was measured every other week in three rooms of each house. In spring and summer, 22 of the 40 homes had readings above 0.1 ppm at one or more times. Those were mainly the UFFI homes and homes aged 5 years or less. Thus, many residences may at times experience levels greater than the 0.1 ppm ASHRAE ceiling (44).

Because the bulk of formaldehyde is emitted in the first year and because conventional homes are not tightly constructed, older conventional homes seldom have formaldehyde problems. Tighter buildings resulting from the energy crisis of the 1970s, however, have lower exchange rates between indoor and outdoor air and generally higher indoor formaldehyde concentrations. Formaldehyde levels of over 0.1 ppm may occur due to low infiltration rates (44).

The best data for exposures in conventional homes is obtained by random sampling of those built after 1982 when lower emission resins came into use. Studies by Lawrence Berkeley Laboratories, CPSC, the Canadian government, and state governments and universities in Iowa, Indiana, Texas and California suggest the average level in conventional homes is currently around 0.05 ppm. The studies found average levels ranging from 0.03-0.09 ppm. Newer and energy efficient homes have levels from 0.1 to 0.2 ppm, while homes over 5 years old average between 0.005 to 0.08 ppm. Comparison of these results with data from studies prior to 1980 indicate little change in conventional home levels (85).

Mobile Homes

Problems with elevated formaldehyde levels have occurred in office trailers, temporary or portable school buildings and mobile homes. Common characteristics of these structures are surface loading factors over 1 square meter of UF bonded products per cubic meter of air, poor insulation and poor ventilation. Such buildings are often located in open areas where sun and

wind can cause the buildings to reach humidity and temperature levels which compound formaldehyde release problems (83).

Mobile homes have the highest formaldehyde exposure levels as compared to all other types of dwellings regardless of age (44). Formaldehyde concentrations were often found to exceed 0.1 ppm in Denmark, the Netherlands, and the Federated Republic of Germany (10). Over 200 mobile homes assessed in Washington state had 0.03 - 2.4 ppm, with similar findings in Wisconsin (7). Median concentrations of 0.1 ppm were found in 100 mobile homes in Texas and 0.58 ppm in Minnesota units where residents complained (83).

Mobile homes built after 1982 were also assessed in the studies by Lawrence Berkeley Laboratories and others discussed above. Mobile home levels have decreased since lower emission products have been in use. If complaint homes are included, levels in individual homes range from less than 0.1 to 1.0 ppm. Average levels are now 0.2 - 0.5 ppm (85).

UFFI Homes

After mobile homes, the highest formaldehyde concentrations are found in homes with UFFI, again regardless of age. UFFI homes have been found to contain levels four times as high as their conventional counterparts, which seldom have high levels. Use of UFFI increased throughout the 1970s. UFFI was installed in 435,000 residences between 1975 and 1981 and was estimated to be in 500,000 American homes by 1983 (44, 86).

Data collected by Cohn et al. from many studies of UFFI homes show that formaldehyde levels decrease as the UFFI ages. Even after 9 years, however, the average reading is above 0.1 ppm (Table 4-4). In this study each of the 1164 points represent the average reading in a UFFI home; most measurements were conducted using the chromotropoic acid method. The most rapid decline was in the first 40 weeks. Other studies have indicated the range of concentrations of formaldehyde in UFFI homes to be from 0.01 to 4.1 ppm with an average of 0.14 ppm (85).

Occupants' Living Habits

Smokers expose themselves and others to formaldehyde produced by combustion. A smoker of a pack of cigarettes a day is exposed to 0.38 mg of formaldehyde. Nonsmokers are also exposed to this formaldehyde (44).

The occupants' living patterns and ventilation control indoor air levels of formaldehyde from tobacco smoking. In studies where 10 cigarettes were smoked in a home each day, formaldehyde levels did not increase significantly over controls. Studies where a number of people chain smoked in a poorly ventilated room showed quick increases in formaldehyde levels (85). For example, in a study where 5 people smoked a total of 20 cigarettes within 30 minutes in an average size room the formaldehyde level rose from 0.01 to 0.27 ppm. Undiluted cigarette smoke contains up to 40 ppm by volume of formaldehyde (44).

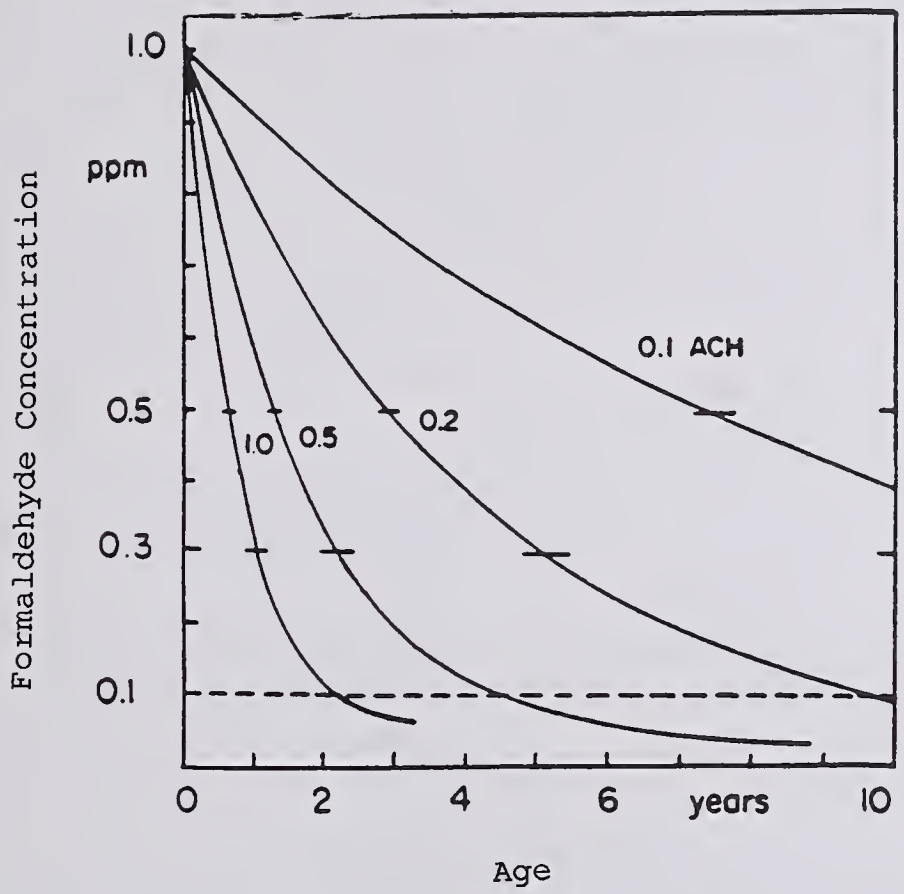
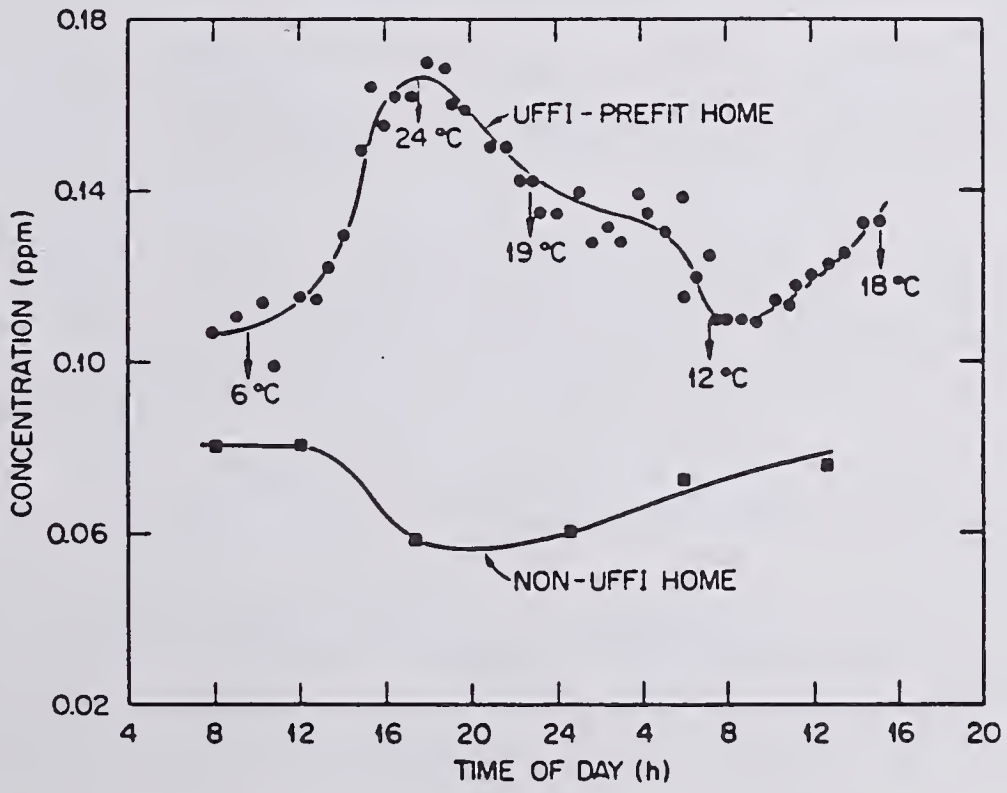


Figure 4-6.
Formaldehyde Release from Particleboard as a Function of Age and Ventilation Rate.

(Source: Meyer, B. and Hermanns, K. Journal of the Air Pollution Control Association, 1985)

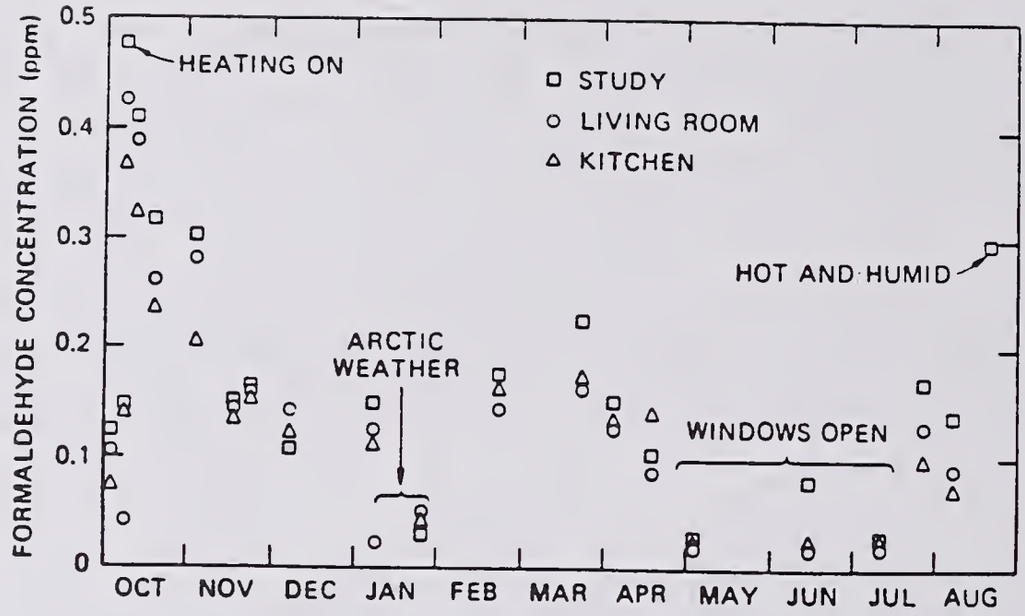
Figure 4-7
Diurnal Variation
of Formaldehyde Concentration
in UFFI and Non-UFFI Homes



(Source: Meyer, B. and Hermanns, K. Journal of the Air Pollution Control Association, 1985)

Figure 4-8.

Variation of Formaldehyde Concentration with Time of Year



The interactive effects of emission and ventilation factors explain the high levels of formaldehyde found in new, tightly-constructed homes (high emission rates of new materials, little ventilation); mobile homes which have a large amount of bonded wood (high emission materials, high load factor, little ventilation due to their tightness); and UFFI homes (high emission of porous material, high load factor).

FORMALDEHYDE: SCOPE OF THE PROBLEM

Rural outdoor air generally contains 0.005-0.05 ppm of formaldehyde. Levels in urban air are almost always below 0.05 ppm and indoor levels are usually below 0.1 ppm (83). Formaldehyde levels may be elevated indoors from the use of formaldehyde building materials, household furnishings or the occupants' habits.

Laboratories and Industrial Facilities

High formaldehyde levels have been found in specialized situations such as laboratories and industrial facilities where formaldehyde is used as part of a process or as a component of a product. For example, in high school and college biology laboratories where specimens are preserved with formalin,

Table 4-4

Formaldehyde Concentration in UFFI Homes

Days	Number of data points	Avg ppm
0-71	63	0.210
141-210	76	0.240
211-280	51	0.240
281-350	58	0.058
351-420	72	0.068
421-490	55	0.084
491-560	68	0.100
561-630	45	0.076
631-700	49	0.078
701-770	70	0.080
771-840	37	0.081
841-910	54	0.079
911-980	45	0.058
981-1050	44	0.082
1051-1120	66	0.072
1121-1190	30	0.050
1191-1260	46	0.040
1261-1330	29	0.072
1331-1400	22	0.054
1401-1470	22	0.074
1471-1540	8	0.063
1541-1610	6	0.047
1611-1680	15	0.032
1681-1750	9	0.021
1751-1820	14	0.067
1821-1890	4	0.102
1891-1960	5	0.039
1961-2030	4	0.080
2031-2100	5	0.054
2101-2170	4	0.050
2171-2240	4	0.078
2241-2310	1	0.040
2521-2590	1	0.117
3011-3080	2	0.030

(Source: EPA's Final Report: "Formaldehyde Exposure in Residential Settings: Sources, Levels, and Effectiveness of Control Options," 1986)

Stoves and heaters are also significant emitters as discussed in the previous section. Clearly, formaldehyde levels above 0.1 are occurring today not only in mobile homes and UFFI homes, but also in new conventional and energy efficient homes and may occur in any home where heavy smoking is occurring.

Formaldehyde Risk Characterization

The risk of adverse health effects is related to the nature of the product present, its formaldehyde release characteristics and environmental factors (83). Assuming that animal data can be extrapolated to humans, the risk of cancer for a given individual is small. For example, the additional risk after 9 years in a UFFI home is estimated at about 1 in 20,000. Individual risk will vary with actual formaldehyde levels and the length of time a person has been living in a home, since as foam ages formaldehyde emissions decrease.

EPA's most recent assessment of formaldehyde's health effects classifies it as a Group B1 probable human carcinogen, based on "sufficient" evidence from animal studies and "limited" evidence from human studies (87).

FORMALDEHYDE MITIGATION

Mitigation of high indoor airborne formaldehyde concentrations is approached through elimination of the emission source, alterations or design improvements to products which reduce emissions, or through removal of formaldehyde from the air. While most mitigating techniques decrease formaldehyde levels, their success varies. Their long term effectiveness and effect on mitigating adverse health effects are unknown. Approaches vary in cost, effectiveness and ease of implementation. They may be used alone or in combination (44).

Source Control

Source elimination is theoretically an effective means of mitigation but may be costly, difficult to achieve, or lack feasibility. UFFI removal involves tearing down interior or exterior walls. Replacing bonded wood products in floors, walls, or built-in cabinets requires major construction work. Replacing furniture can be a major expense. In many cases substitute products are not available or practical.

Techniques which have been used to reduce formaldehyde levels through emission control include emission barriers, chemical treatment, change in product composition, and treatment at the factory. Sealing interior wall entry points has been used in UFFI homes. Ammonia has been sprayed inside

both UFFI and non-UFFI houses to lower formaldehyde concentrations. Painting or otherwise coating an object can retard emission. Some vapor barrier paints are manufactured (44).

Emissions from biological specimens can be reduced by rinsing followed by immersion in a low or no formaldehyde medium. This treatment has reduced formaldehyde levels in laboratories several fold. Time may also be regarded as a mitigant since the most reactive components of a product that generates and emits formaldehyde are usually "exhausted" first. Decreased levels over time have been found in both UFFI and non-UFFI homes (44).

Elevated indoor formaldehyde levels may be due to defective or improperly installed products such as a single panel of raw bonded wood or a poorly vented stove. Mitigation is straightforward in these cases.

Compressed Wood Products

Emissions have been reduced by changing resin composition, adding scavengers to the wood before pressing, adding urea or wax solutions to the wood finish, fumigating with ammonia while the wood is still hot, and other methods (83). Since there are two types of emissions, long term hydrolysis and decomposition and short term release of free formaldehyde, controls may reduce one or both types and sometimes decrease one while increasing the other.

Low formaldehyde resins have now decreased the residual formaldehyde content of products by a factor of 10 (83). During the 1970s resin was often produced using a 2:1 ratio of formaldehyde to urea. This has now been reduced to around 1.2 to 1.5:1 through use of different resins and has led to a decline in emissions because less excess formaldehyde is present. In the early 1960s when the first conferences on formaldehyde reduction occurred in Germany, some resins contained 1-6% free formaldehyde by weight. This percentage has been reduced by a factor of as much as one million (83, 85).

PF emits relatively small amounts of formaldehyde and isocyanate resins contain none (85). These resins are of minor concern.

Emission barriers both reduce emission rate from the pressed wood and reduce the amount of water the product absorbs. Scavengers which combine with and bind the free formaldehyde are being added to UF resin-wood systems. These decrease short term emissions but their effect on long term emission is uncertain. Some coatings have scavengers which react with formaldehyde. Spraying the surface with a scavenger or exposing it to reactive gaseous ammonia has been reported to reduce early emissions by up to a factor of 10. Painting wood surfaces, adding other coatings or vinyl veneers and decorative overlays inhibit emissions to varying degrees. Some paints, for example, prevent 98% of emissions and some wallpapers prevent around 30% (85). Gypsum board and carpets also act as barriers (83) although gypsum may lead to other indoor air problems.

Before low emission products were in common use, chemical remedial methods such as ammonia fumigation were used. Ammonia reaches most of the emitters except combustion sources (92) but results are often unreliable (83).

By mid-1984 50% of the UF bonded wood products made in the United States met HUD requirements for mobile homes.

UFFI

The National Research Council of Canada has recommended a technique to lower formaldehyde levels in UFFI homes. Walls should be sealed by repairing all holes, cracks or gaps with caulking or spackling compounds and applying two coats of vapor-barrier paints or mylar, vinyl or good grade canvas backed wallpaper (86). However, painting and papering may not be enough to keep formaldehyde from entering living areas. Junctions of walls and floors should be caulked with butyl or acrylic latex sealants or sealed with weather stripping or foam-backed tape. Applying a sealant such as varnish to joints and surfaces may help (86).

High concentrations of ammonia may be pumped into the house. This can be hazardous and should only be done by trained personnel. It is unclear whether low ammonia concentrations are effective. Ammonia also corrodes brass electrical and gas fittings and connectors (86).

Canada considers removal a costly method of last resort. There have been some cases where UFFI was removed but health effects have persisted, possibly due to improper removal (86). The removal procedure calls for removing interior or exterior wall panels or siding, removal of the UFFI, and addition of new insulation and siding. Any wood surfaces which were in contact with the UFFI are treated with a stabilizing agent such as sodium bisulfite.

In fifteen Wisconsin homes variously treated by sealing or removing particleboard and venting or removing UFFI, formaldehyde levels decreased significantly compared to the levels in fifteen untreated houses (44).

Removal

Increased ventilation is the only mitigation technique that is not an emission control (85). As a result of the complex interaction between formaldehyde emission rates and airborne formaldehyde concentration, ventilation is not as effective in reducing concentration on a short term basis as it is with other pollutants.

Formaldehyde emission rates are determined by the difference in pressure or concentration between the emitting product and the indoor air. Ventilation lowers the airborne formaldehyde concentration. This increases the pressure difference leading to greater formaldehyde emissions.

As a result of the increased emission or "outgassing," the dilution curve for formaldehyde indicates a reduction in airborne concentration of 33-38% rather than 50% for every doubling of ventilation rate (ach). It should be noted that since a product contains a finite amount of formaldehyde, increasing the ventilation rate accelerates the aging process.

Levels of formaldehyde have been reduced from 0.5 to 0.1 ppm by filtering air of mobile homes through a bed of alumina pellets impregnated with permanganate. A heat pump in its cooling mode may be effective since moisture condensing onto and water drying from the cooling coils may remove formaldehyde vapor. Forced air ventilation or air cleaners may be used. The effectiveness of portable air cleaners and the lifetimes of their "sorbent" cartridges have not been tested (44).

In moderate climates where heating and cooling of homes are intermittent, there may be incomplete air mixing leading to pockets of high formaldehyde concentration. Examples are paneled rooms, display shelves and areas with cabinets such as kitchens recently remodeled. In residences or commercial buildings with forced air heating or air conditioning and especially in severe climates where these systems are in use much of the time, air mixes and pockets of high formaldehyde are less likely to occur (83).

NASA is conducting research regarding removal of pollutants from indoor air. Chamber studies have been conducted with more than a dozen common houseplants that may be able to metabolize formaldehyde. High levels of formaldehyde were introduced, and after 24 hours, all plants reduced formaldehyde levels in the chambers. The elephant ear philodendron, golden pothos and spider plant each removed at least 80% of the formaldehyde present and the aloe vera 90% (93).

FORMALDEHYDE: FEDERAL INITIATIVES

Federal action to reduce indoor formaldehyde levels has taken the form of air quality and product emission standards and, in the case of UFFI, a ban on sales.

Air Quality Standards

OSHA considers formaldehyde a potential occupational carcinogen. Over 200,000 workers are estimated to be exposed in laboratories, the funeral service and apparel industries (94).

OSHA in 1985 announced its intent to lower its 3 ppm workplace standard for airborne formaldehyde to 1 or 1.5 ppm. NIOSH recommended that OSHA's 3 ppm 8-hour time weighted average be reduced to 1 ppm (84). In late 1987 OSHA reduced the 3 ppm standard to 1 ppm (94).

A number of standards have been developed based on formaldehyde's irritant nature. Carcinogenic effects of formaldehyde exposure in animals has not been a major consideration. ASHRAE has adopted a guideline of 0.1 ppm for indoor air which it suggests provides a comfortable environment for most exposed people, but this is not a guarantee of health protection, especially for sensitive individuals (44). The American Industrial Hygiene Association

(AIHA) also recommends a 0.1 ppm guideline. In 1978 the Netherlands established 0.1 ppm as an indoor standard for maximum permissible concentration. West Germany, Sweden, and Denmark are considering similar standards (84). However, NAS has reached the conclusion that the vast majority of healthy adults would not suffer symptoms of irritation at 0.25 ppm (5).

Product Standards

In May 1984 EPA initiated a priority regulatory investigation of formaldehyde under the Toxic Substances Control Act (TSCA), focusing on resins used in pressed wood construction materials and formaldehyde in permanent press clothing (87).

HUD has no emission standard for formaldehyde products used in houses in general (5). However, in August 1984 HUD changed its Manufactured Home Construction and Safety Standards to limit formaldehyde emissions in mobile homes. This is a product standard which limits the level of formaldehyde from interior plywood, particleboard, floor decking and cabinets. Emissions must not exceed 0.2 ppm from plywood or 0.3 ppm from particleboard in a chamber test. Products must be certified as meeting the standard. Ambient levels in manufactured homes built with such materials are not expected to exceed 0.4 ppm at 77 degrees Fahrenheit, 50% relative humidity and 0.5 ach of outdoor ventilation. This is roughly twice the actual average measured rate for new mobile homes (87).

UFFI Ban

CPSC began an investigation of formaldehyde use in consumer products in 1978. Because formaldehyde emissions are uncontrollable and potentially carcinogenic, CPSC banned UFFI in schools and residences in 1982. Canada had banned UFFI sales late in 1980 (44, 87).

After the UFFI ban was issued, a number of lawsuits were filed asking that it be modified or set aside. In April 1983 an opinion was issued by the U.S. Fifth Circuit Court of Appeals to set aside the ban and therefore allow sales of UFFI. Its reasoning was that the CIIT study and the home measurement and laboratory test data used to estimate formaldehyde levels in UFFI homes were not an adequate basis for estimating cancer risk. CPSC was also asked to quantify the severity of acute symptoms of UFFI exposure and the number of persons affected. CPSC countered with a petition for a rehearing indicating that the New York University study confirmed the CIIT results, its lab tests were valid and its measurements of levels in homes were "for all intents and purposes," a random sample. A second opinion in June 1983 corrected some factual errors but denied the petition (86).

UFFI has virtually disappeared from the commercial market in the U.S. and Canada (83). CPSC is investigating formaldehyde emitting products and

pursuing national consensus standards reducing emissions in products made from pressed wood (87).

FORMALDEHYDE: MASSACHUSETTS INITIATIVES

Between 1970 and 1981 UFFI was installed in about 7000 homes in Massachusetts. DPH banned UFFI in November 1979 because of many documented acute health problems. At the time of the ban, DPH developed a repurchasing program with regulations requiring UFFI removal at industry expense. The formaldehyde industry challenged the regulations in court. The DPH regulations were found to be valid with a few minor changes.

In 1985, with the cooperation of DPH and industry, UFFI legislation was passed. Chapter 728 of the Acts of 1985 (effective July 1986):

- Established an air testing program for any homeowner with UFFI installed prior to December 31, 1980.

- Established a removal program for homes where levels of formaldehyde are greater than 0.1 ppm or an occupant has experienced adverse health effects and meets certain documentation criteria.

- Established a trust fund supported by contributions from industry members to pay for air testing and removal. Those who receive payment for UFFI removal cannot take legal action against any industry member who has contributed to the fund. Industry members who make "significant" contribution to the fund are exempt from the repurchase regulations.

- Directed DPH to promulgate regulations regarding approval of testing labs, removal methods and approval of removal contractors. These regulations (105 CMR 651.000) became effective in December 1986.

- Established a UFFI hotline (toll free 1-800-222-UFFI).

- Determined that homeowners, landlords, bankers and realtors are not liable for UFFI-related health problems if disclosure is made by seller to buyer or landlord to tenant. Sellers and landlords must determine whether the house has UFFI. Banks and realtors cannot discriminate against homes with 0.1 ppm of formaldehyde or less.

Massachusetts has established a 0.1 ppm action level, though it recognizes homes with concentrations well below 0.1 may adversely affect occupants. DPH and DEQE believe this level should be lower.

The air testing protocol involves hanging vials in two rooms of the house, other than the kitchen or bathroom, where levels would be predictably higher. They remain in place for 7 days then are returned to DPH for analysis. If the reading is over 0.75 ppm, further testing is done. The initial test costs $13 for the kit and $22 for analysis. The second test

takes 90 minutes and costs $150-$240 depending upon the location of the home. Currently analysis is done out of state since laboratories are selected based on competitive bidding. About 2.5% of the homes test above 0.1 ppm with an average level of 0.05 ppm.

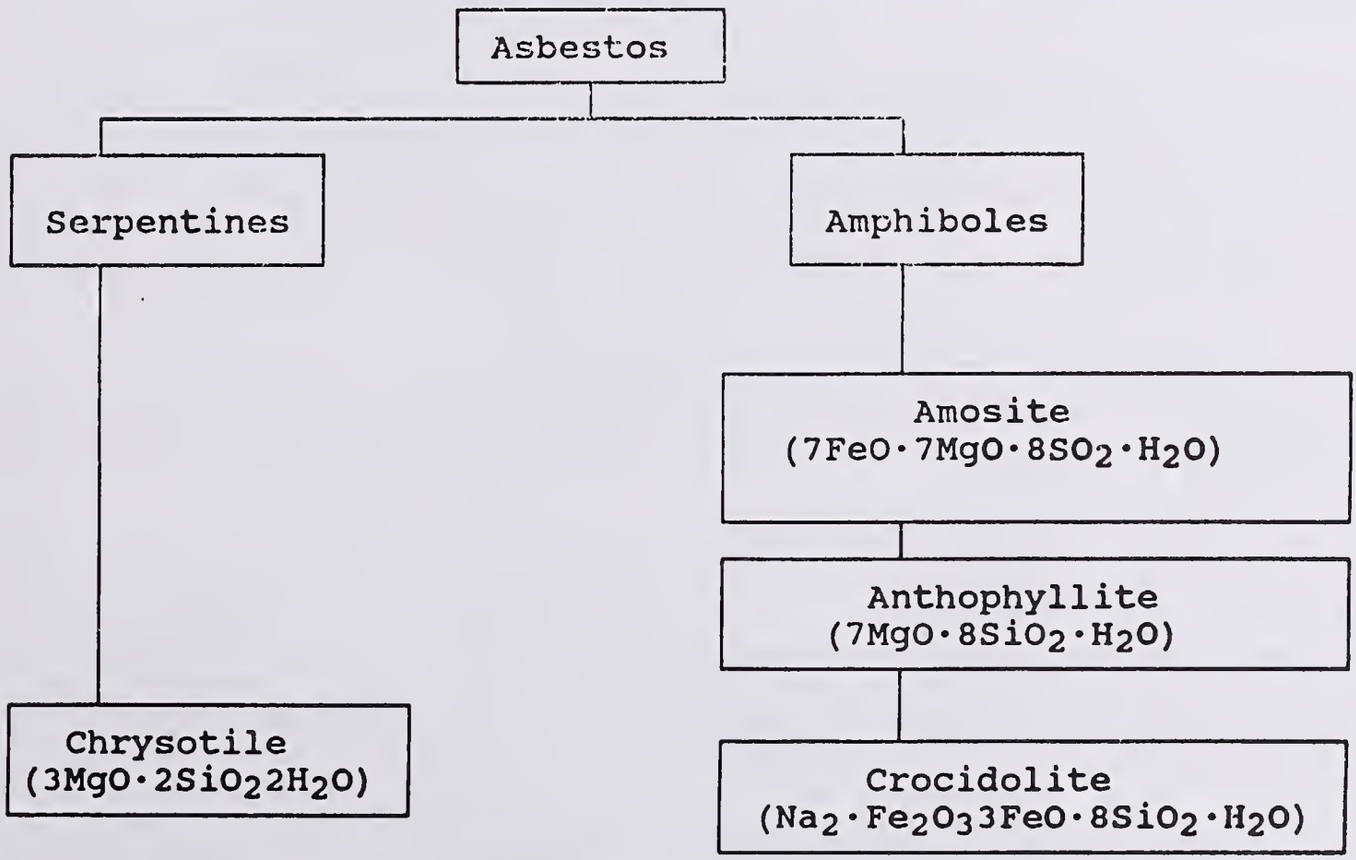
Chapter 5: ASBESTOS

ASBESTOS AND ITS PROPERTIES

Asbestos is a generic term for a group of naturally occurring hydrated mineral silicates, belonging to two mineral groups: the amphiboles, such as amosite, crocidolite, tremolite, anthophyllite, and actinolite, as well as the serpentines, including the most commonly used type of asbestos, chrysotile (See Figure 5-1). All of these silicate minerals occur naturally in both a fibrous and non-fibrous form. Federal and state laws, however, regulate only the fibrous, or asbestiform, varieties.

FIGURE 5-1

Mineralogic Classification and Chemical Composition of Common Commercial Types of Asbestos



(Source: Adapted from The New England Journal of Medicine, 1982, P.306)

Asbestos fibers owe their commercially valued properties of strength, flexibility and physical and chemical durability largely to their fibrous structure. The strength and flexibility of asbestos fibers is diameter-dependent; the smaller the diameter of the fiber, the greater its strength. Asbestos fibers are characterized by an extreme length to width ratio. They have smooth, parallel longitudinal faces, which may potentially be responsible for the high strength of the surface layer of asbestos fibers. (95) Asbestos fibers are soft and flexible enough to be spun into thread and woven into cloth. Asbestos will not burn although it disintegrates at extremely high temperatures and it is resistant to chemicals.

Chrysotile fibers account for most of the asbestos use in the United States. Its fibers may be as small as 0.015 microns in diameter; thirty thousand feet of thread can be spun from one pound of these fibers. Chrysotile has a tensile strength equal to that of good grades of steel wire and can withstand temperatures up to 700 degrees Fahrenheit. (96)

Commercially valuable but less often used are the amphibole fibers, including various silicates of magnesium, iron, and calcium. These fibers are generally brittle and cannot be spun like chrysotile, however, they are more resistant to heat than chrysotile fibers.

HISTORIC USE OF ASBESTOS

Asbestos fibers provide a unique conglomerate of physical and chemical properties, the utility of which has been recognized since the days of the early Roman Empire. Plutarch, a Roman historian, described asbestos lamp wicks. During the Dark Ages, the Emperor Charlemagne is said to have awed warrior guests from a rival kingdom by throwing an asbestos tablecloth into the fire to "clean" it, then withdrawing it, intact and unsinged from the flames. (97)

In the nineteenth century, Henry Ward Johns of Quebec mixed asbestos with burlap, pitch and manila paper, developing the first asbestos roofing product. Following his death, Mr. Johns' company was purchased by C.B. Manville to form the Johns-Manville Company.

The use of asbestos grew dramatically in the twentieth century. (See Table 5-1) Its commercially significant combination of properties - high tensile strength, flexibility, durability, resistance to fire, heat and corrosion - has resulted in the ubiquitous use of asbestos primarily in the construction industry. During World War II asbestos was used widely in the shipbuilding industry to insulate boiler and piping systems. The list of uses grew even longer with asbestos being used as a fire retardant and in building components, as an insulator from heat, cold and electricity, in acoustical tiles, brake linings, clutch facings, gaskets, roofing, paint fillers, chemical filters, as a reinforcing agent in cement and vinyl products, and as an adhesive agent in asphalt. (87, 98)

TABLE 5-1

Consumption of Asbestos in the United States

Product	Average percent asbestos	Binder	Dates used
Friction products	50	Various polymers	1910-present
Plastic products			
Floor tile and sheet	20	PVC, asphalt	1950-present
Coatings and sealants	10	Asphalt	1900-present
Rigid plastics	<50	Phenolic resin	?-present
Cement pipe and sheet	20	Portland cement	1930-present
Paper products			
Roofing felt	15	Asphalt	1910-present
Gaskets	80	Various polymers	?-present
Corrugated paper pipe wrap	80	Starches, sodium silicate	1910-present
Other paper	80	Polymers, starches, silicates	1910-present
Textile products	90	Cotton, wool	1910-present
Insulating and decorative products			
Sprayed coating	50	Portland cement, silicates, organic binders	1935-1978
Trowelled coating	70	Portland cement, silicates	1935-1978
Preformed pipe wrap	50	Magnesium carbonate, calcium silicate	1926-1975
Insulation board	30	Silicates	Unknown
Boiler insulation	10	Magnesium carbonate, calcium silicate	1890-1978
Other uses	<50	Many types	1900-present

(EPA's Asbestos Waste Management Guidance, 1985)

Some of these applications of asbestos containing products result in airborne asbestos fibers, for example, sprayed-on fireproofing insulation, acoustical plasters, ceiling tiles, and pipe and boiler insulation. Asbestos fibers may become airborne due to mechanical disturbances, water damage, or other disruptive forces.

Although identified by European studies much earlier, it was not until the 1960s and early 1970s that the relationship of asbestos to lung cancer and mesothelioma was demonstrated in various U.S. health studies. The publication of these studies resulted in the U.S. Environmental Protection Agency ("EPA")

instituting a ban in 1973 on the spraying of fire proofing and insulating materials containing greater than one percent asbestos by weight. Later in 1973, the EPA banned all sprayed materials containing more than one percent asbestos.

The Consumer Product Safety Commission (CPSC) has banned the use of asbestos textiles in general use garments, in artificial fireplace materials and asbestos containing spackling and taping compounds. (99)

A CPSC program identifies household appliances which could release asbestos fibers during use. The general reduction in use and increased caution in placement of asbestos in these appliances in recent years makes it unlikely that asbestos components present a significant health risk from release of asbestos fibers. (95) In 1979 manufacturers voluntarily recalled hair dryers containing asbestos heat shields; current models do not contain these shields. (95)

Today, asbestos is still used for numerous purposes, including gaskets, packing, and brake linings, and as a cement reinforcing agent. While no single substance has been found with all of the properties of asbestos, some alternatives to asbestos have been identified and used either alone or in combination with other substances to replace asbestos in some of its previous applications. Whether the same health issues will arise with such asbestos substitutes remains largely unknown.

ASBESTOS AND PUBLIC HEALTH

Most of the epidemiological information available on asbestos involves occupational exposure to asbestos. Generally, asbestos dispersed in the air or drinking water is viewed as the health threat. Cases of asbestosis were first identified in the late 1800s in asbestos miners and others working with asbestos. In 1935, asbestos was first correlated with lung cancer, and later in the 1950s with the extremely rare cancer called mesothelioma. In order to assess the risks to the general public associated with non-occupational exposure to asbestos, epidemiologic data from occupational exposure must sometimes be extrapolated.

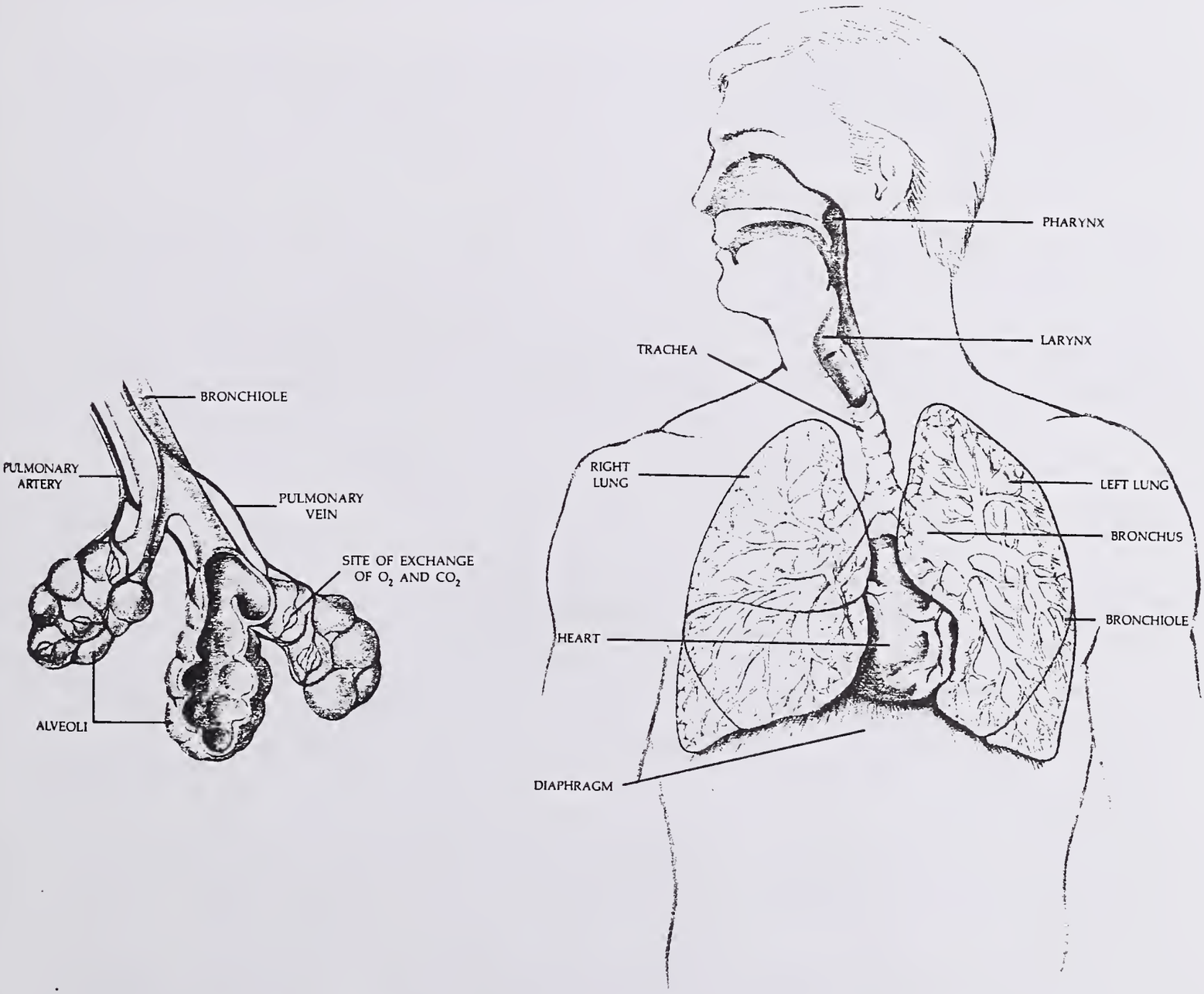
Distribution and Deposition of Asbestos in the Human Respiratory System

Before the impact of asbestos on human health can be comprehended, an elementary understanding of the human respiratory system is necessary. The human respiratory system allows us to breathe in oxygen needed by each cell in our bodies to function properly and to exhale carbon dioxide, a waste product of human metabolic processes. This exchange occurs through the inflow and outflow of air between the atmosphere and the lung alveoli called pulmonary ventilation. It is in the alveoli, the terminal portions of the lungs, that this critical gas exchange occurs. Thin capillary and alveolar membranes

accommodate the diffusion of oxygen and carbon dioxide. See Figure 5-2 for a detailed drawing of the respiratory system components.

FIGURE 5-2

Human Respiratory System Diagram



(Source: Curtis, Helena. Biology(Fourth Edition), Worth Publishers,Inc. 1983)

The process of breathing entails the alternating compression and distension of the lungs thus causing the pressure in the alveoli to rise and fall. During inspiration, a slightly negative pressure occurs in the alveoli drawing air into the lungs. The opposite occurs during expiration.

The respiratory system has the capacity for clearing itself of foreign particles, such as some types of asbestos fibers. This feat is accomplished through a number of measures. The first line of defense begins in the nasal cavity where tiny hairs called vibrissae filter out particles greater than 10 microns in diameter. The mucoid lining of the sinuses also traps larger particles. In the trachea and bronchi, a sticky mucus blanket constantly moves upward, propelling foreign particles to the throat where the mucus is either swallowed or expectorated.

Fibers up to 200 microns in length may pass through the respiratory system's clearing mechanisms, provided the diameter does not exceed 3 microns. (99) Size and other morphological characteristics ultimately dictate whether and to what extent deposition of asbestos fibers occurs in the lungs.

Particles that reach the membrane surfaces of the alveoli will likely be attacked by alveolar macrophage cells, part of the bodies defense system. These cells engulf and digest foreign particles. Sometimes asbestos fibers are found in the lung, coated with proteinaceous compounds. In this form, the fibers are called asbestos bodies and are believed to result from macrophage activity. Some asbestos fibers will inevitably get through the body's defense mechanisms. The major resultant health effect is fibrosis of the lung, a process by which healthy lung tissue is replaced with scar tissue.

Asbestosis

Exposure to asbestos may result in asbestosis, or pulmonary fibrosis. Extensive epidemiologic evidence of asbestosis has been documented in the textile, insulation, and shipbuilding industries.

The process of pulmonary fibrosis involves the replacement of healthy functional lung tissue with fibrous connective, or scar, tissue. The presence of such scar tissue in the lungs impedes the gaseous exchange of oxygen and carbon dioxide between the alveoli of the lungs and surrounding blood capillaries due to blockage and the loss of elasticity needed for the inhalation/exhalation function. (101) Scar tissue impairs lung capacity resulting in the dyspnea, or shortness of breath, experienced by victims of asbestosis. Dyspnea is generally the first clinical sign of asbestosis, followed by rales and clubbing or thickening of the fingers. (96) Extensive damage can result eventually in respiratory or cardiac failure. (102)

While the earliest cases of asbestosis were noted in the late 1800s it was not until 1924 that asbestosis was given its name by Dr. W.E. Cooke in the British Medical Journal, reporting on the case of a young woman who had worked with asbestos and had died with extremely scarred lungs. By the 1930s,

asbestos inhalation was widely established as the cause of asbestosis. (103)

Lung Cancer

A considerable amount of epidemiological evidence points toward a causal relationship between asbestos exposure and the development of lung cancer. Lung cancer is characterized by an increase in intensity and persistance of cough, chest pain to a degree which gives a sense of restriction and possibly anorexia. Asbestosis symptoms often mask the onset of lung cancer since the two conditions result in similar symptoms.

Little is known about the cancer-causing mechanism of asbestos fibers. It has been reported that the shape and size of asbestos fibers and the rate of degradation in the lungs play an important role in the production of cancer. (104) While details of the cancer-causing mechanism are largely unknown, a sufficient amount of evidence exists to establish a need for addressing the lung cancer threat of asbestos.

Synergistic Effect of Asbestos and Smoking

Most existing data support a multiplicative interaction between smoking and asbestos inhalation in the promotion of lung cancer. (105) (106) In 1979, 17,800 insulation workers were studied to determine what if any synergistic relationship existed. The rate of lung cancer mortality was assumed to be 11.3 persons/100,000 persons per year for those who did not smoke or work with asbestos. Of those individuals studied, the rate of lung cancer mortality was 58.4/100,000 for non smokers who worked with asbestos, 122.6/100,000 for smokers who did not work with asbestos, and 601.6/100,000 for those who both smoked and worked with asbestos. (107) Based upon these results, exposure to asbestos increases the lung cancer rate by a factor of five (5) for nonsmokers and by a factor of fifty (50) for smokers relative to unexposed nonsmokers. In a study with rats, Topping and Nettesheim showed that cigarette smoke and asbestos exposure need not occur simultaneously for a synergistic effect to occur. (105)

Mesothelioma

Mesothelioma is characterized by diffuse tumor growth in either the lining surrounding the lungs (the pleura) or the lining of the abdominal cavity (the peritoneum). Symptoms of pleural mesothelioma include dyspnea, pleural effusion, or fluid in the chest cavity, pain in the chest, and progressive weight loss. (105) Death results from pulmonary insufficiency. (108) Peritoneal mesothelioma is characterized by abdominal pain and distension, with the accumulation of fluid in the peritoneal cavity. Death results from pronounced wasting or exhaustion from the inability to assimilate food. (108)

By 1960, asbestos was known as the cause of mesothelioma. One notable study was performed by a South African pathologist, J. Christopher Wagner. Dr. Wagner discovered several dozen cases of this rare and fatal tumor among people whose only exposure to asbestos was their proximity to asbestos mills and dumps. (97) This case represents the first evidence for a definite etiologic link between asbestos and mesothelioma. (108)

The disease hardly ever occurs without some asbestos exposure and for this reason, mesothelioma is considered the "marker" disease for asbestos exposure. (97) In fact, the incidence of mesothelioma has increased dramatically in recent decades, paralleling the increased use of asbestos in industry since 1930. (108) No treatment is available for mesothelioma and it is virtually always fatal within two years of diagnosis. (105)

Gastrointestinal Tract Cancer

Asbestos has been implicated as a potential cause of gastrointestinal cancer in asbestos workers since 1964. (109) It has been suggested that asbestos enters the gastrointestinal tract by drinking contaminated water supplies or by the clearance process of the respiratory system discussed earlier.

Sudden Infant Death Syndrome and Bronchopulmonary Dysplasia

Asbestos bodies in children's lungs have recently been associated with Sudden Infant Death Syndrome (SIDS) and Broncopulmonary Dysplasia (BPD), or faulty development of portions of the respiratory system. The lungs of 10 out of 46 lungs from autopsied children (ages 1 to 27 months) contained asbestos bodies, which are generally considered a marker for asbestos exposure. Seven were diagnosed with SIDS and three with BPD. (110)

This study provides no evidence of a causal relationship between asbestos and SIDS or between asbestos and BPD, only an association. No background study was conducted and no statistical analysis was employed to determine whether any significant relationship exists between these ailments and the occurrence of asbestos bodies. Possibly the asbestos bodies are a symptom of an underlying abnormal lung physiology in which the clearing mechanism is disrupted. Some as yet unknown environmental factor may be the cause, such as excess levels of asbestos in the ambient air or in drinking water. (110)

In summary, further study is needed to clarify whether any relationship exists between asbestos bodies in children's lungs and SIDS and between these bodies and BPD.

Asbestos Risk Characterization

Risk associated with exposure to asbestos can be calculated using mathematical models which translate cumulative exposure into expected numbers of deaths. The EPA conducted such a risk assessment for exposure to asbestos in public and commercial buildings. (111) Risks for mesothelioma and lung cancer were considered, but not for asbestosis which is generally not associated with non-occupational exposure to asbestos.

EPA was unable to obtain reliable estimates of risk given the paucity of data associated with airborne asbestos fiber levels. To compensate for this lack of data, EPA used arbitrary airborne asbestos levels in their "proportional risk" model. The results of the model, therefore, do not represent absolute magnitudes of risk expected in public and commercial buildings, but instead indicate which asbestos exposure source (schools v. public and commercial buildings) contributes more to the total risk. While these results do not provide actual measures of risk, they are useful in establishing priorities for reducing asbestos exposure.

Table 5-2 shows the results of one proportional risk model prepared by EPA. It shows the percent risk attributable to public and commercial buildings for both lung cancer and mesothelioma under various exposure scenarios. The scenarios differ in the relative airborne asbestos levels in school v. public and commercial buildings and in the amount of time spent in public and commercial buildings. The model assumes thirteen (13) years are spent in school with no asbestos exposure occurring in public and commercial buildings during this period.

The results in Table 5-2 demonstrate that early exposure is more important relative to mesothelioma; that is, the incidence of mesothelioma increases with the time from onset of exposure. The incidence of lung cancer depends more upon cumulative exposure, thus in Table 5-2 long post-school exposures dominate the time spent in school in terms of proportional risk for lung cancer. In sum, EPA concluded that exposure in public and commercial buildings contributes more to lung cancer risk than to mesothelioma risk (111).

Risk associated with occupational exposure has been projected for the period of 1985 to 2009, estimating the incidence of asbestos-related disease mortality in the United States. The projections are as follows: 21,500 cases of mesothelioma, 76,700 cases of lung cancer and 33,000 cases of gastrointestinal cancer, for a total of 131,200 asbestos-related deaths in this twenty-five (25) year period. (112)

TABLE 5-2 THE PROPORTION OF LIFETIME RISK ATTRIBUTABLE TO EXPOSURE TO AIRBORNE ASBESTOS IN PUBLIC AND COMMERCIAL BUILDINGS RELATIVE TO TOTAL RISK FROM EXPOSURE IN BUILDINGS FOLLOWING 13 YEARS OF EXPOSURE IN SCHOOLS

Exposure Experience	Airborne Asbestos Levels in Public and Commercial Buildings Relative to Levels in Schools				
	Twice	Same	One half	One sixth	One tenth
	Percent Risk Attributable to Public and Commercial Buildings				
Mesothelioma					
13 years at school plus:					
lifetime in other buildings	61	43	28	11	7
20 years in other buildings	61	43	28	11	7
10 years in other buildings	47	31	18	7	4
5 years in other buildings	31	19	10	4	2
Lung Cancer					
13 years at school plus:					
lifetime in other buildings	87	76	62	35	24
20 years in other buildings	75	60	43	20	13
10 years in other buildings	61	44	28	12	7
5 years in other buildings	44	28	16	6	4

(U.S. EPA Study of Asbestos- Containing Materials in Public Buildings, February 1988)

STATE INITIATIVES

Legislative Commission on Asbestos

Concern about the potential health hazard posed by asbestos in public buildings and schools resulted in the establishment of a Legislative Commission on Asbestos in 1975. The Commission's mandate called for the investigation and evaluation of the public health hazard of asbestos in schools and public buildings of the Commonwealth.

Specific goals of the school investigation program included the following:

1) Identify schools where asbestos had been used as a "spray-on" material;

2) Determine the extent of asbestos contamination in these buildings;

3) Propose methods of asbestos containment and removal; and

4) Determine the economic impact of such corrective procedures. (11)

The Division of Occupational Hygiene (DOH) within the Massachusetts Department of Labor and Industries (DLI) implemented the investigation to satisfy these goals. This investigation, however, was limited only to "sprayed-on" forms of asbestos since it was assumed that this material represented the greatest health threat. Out of 1400 public schools built between 1946 and 1972, 178 contained an asbestos sprayed-on material, excluding any non-public areas in the buildings.

By 1978, most schools had been surveyed, and the Commission then notified all school districts as to the survey results and the Commission's remediation recommendations. Compliance with Commission recommendations was voluntary.

While the Commission's work continued, in the late 1970s, the Division of Air Quality Control of the Department of Environmental Quality Engineering began asbestos control efforts to counter the air pollution caused by construction projects that renovated (or demolished) heating and building components wrapped in or containing asbestos. The air program inspections, work practice standards for demolition and renovation, and the designation of asbestos demolition materials as a "special waste" helped to ensure that waste asbestos materials would not become airborne and pose a threat to public health. Late in the mid 1980s, Massachusetts accepted formal delegation of the federal asbestos program and developed its own, more stringent, specific regulation enhancements.

After two years of strictly surveying schools, the Asbestos Commission turned to the issue of asbestos in public buildings. The public building survey differed from the school survey in that all areas of the building were examined, not just areas open to the public.

In its Final Report entitled "1982 Annual Report of the Special Commission Relative to Evaluating the Extent of the Use of Asbestos in the Schools and Public Buildings of the Commonwealth," the Asbestos Commission summarized its activities and made recommendations. The Commission worked hard to heighten public awareness of asbestos hazards through its surveys. A major recommendation of the Commission called for its placement in a permanent state agency to continue its oversight of potential asbestos health hazards.

Chapter 614 of the Acts of 1986

Several recommendations of the 1982 report were included in Chapter 614 which authorized a grant program to reimburse municipalities and school districts for 60-75% of the cost to remove, encapsulate or enclose asbestos found in public schools. Only work contracted before June 30, 1989 is eligible for reimbursement. A total of $30 million was appropriated for asbestos abatement: $25 million in grants and $5 million for an asbestos abatement program for private elementary and secondary schools. (113)

As of the end of October 1988, the Asbestos Program of the DOH still had about 10 million dollars in grant monies available, primarily due to the delay in implementing asbestos abatement projects due to new federal asbestos abatement requirements under the Asbestos Hazard Emergency Response Act (AHERA). AHERA requires that schools be inspected for the presence of asbestos containing materials, especially where asbestos fibers may become airborne, and that an Asbestos Management Plan be prepared prior to the actual abatement of asbestos for which state reimbursement funds are available. Now that the October 12, 1988 AHERA-established deadline for submittal of Management Plans has passed, communities have a better idea what resources they need to spend for abatement and therefore for what amount they should request reimbursement. (114) As of October 12, 1988, the Asbestos Program had received 325 applications for reimbursement, including approximately 800 Management Plans prepared pursuant to AHERA.

In a report to the Massachusetts House Post Audit and Oversight Committee, Jeffrey W. Eiseman, Ph.D. points out an inequity of Chapter 614 relative to reimbursement of public versus private schools. Public schools must raise and appropriate the entire amount of abatement costs, pay the contractor and then apply for reimbursement. Private schools, on the other hand, need only raise their final share of the asbestos abatement costs from 25-40%, and the state pays its share directly to the contractor. This method avoids the prohibition against state funding going directly to private schools, yet it results in a substantial inequity in the treatment of public schools. Dr. Eiseman suggests that this inequity would be remedied if public schools also were required to raise and appropriate only their final share of abatement costs. (115)

A second criticism of Chapter 614's implementation involves the formula used for calculating percentage reimbursement levels. Dr. Eiseman believes the formula would be more equitable if the reimbursement percentage was a

function of the hardship the asbestos abatement project represents to a specific district. (115)

Asbestos Interagency Task Force

In January 1985 the Asbestos Interagency Task Force was formed to formulate and coordinate a comprehensive plan to address the problem of asbestos in public buildings of the Commonwealth. The Task Force issued its recommendations in November 1985; these recommendations were largely reflected in the Governor's Budget Recommendation for Fiscal Year 1987. (116)

Some of the Task Force's more significant recommendations included:

(1) Passage of legislation that would enable DLI to prepare regulations for the removal, containment or encapsulation of asbestos and which would require licensing of abatement workers so that safe work practices are enforced for all asbestos abatement work. Such legislation was passed and DLI promulgated regulations which became effective October 30, 1987. These regulations are described in greater detail below.

(2) Investigation of options for ensuring the availability of long term worker compensation and liability insurance to asbestos abatement contractors and asbestos disposal areas.

(3) Education of state employees about the health threats of asbestos and safe work practices. (117)

STATE REGULATION OF ASBESTOS

Summary of State Authority

There are three (3) state agencies in Massachusetts which play a significant role in the regulation of asbestos projects: The Department of Environmental Quality Engineering (DEQE); the Department of Labor and Industries (DLI); and the Department of Public Health (DPH). Within the past year and a half both DEQE and DLI have amended or promulgated their regulations pertaining to asbestos. DEQE's asbestos regulation as amended in December 1987 requires notification and strict work practices for all size abatement jobs. When DLI promulgated its regulations on October 30, 1987, Massachusetts became one of thirty-two states to have specific requirements for the licensing and certification of asbestos abatement contractors, consultants, workers, and laboratories.

STATE PROGRAMS

DEQE

DAQC (Division of Air Quality Control) enforces NESHAPs standards; regulates visible emissions of asbestos; requires notifications for removal, storage, transport and handling; inspects demolition/renovation and manufacturing operations;
Regulations: 310 CMR 7.00, 7.09(5), 7.15

DSW (Division of Solid Waste) regulates storage, handling, and landfilling of asbestos and asbestos-containing material;
Regulations: 310 CMR 18.00 (transfer stations), 19.00

DHW (Division of Hazardous Waste) regulates hazardous material clean-up; material must be reported if released to the ambient air; friable asbestos is a hazardous material if not properly contained;

RTK (Right to Know) regulates filing of Material Safety Data Sheets for asbestos in the workplace;
Regulations: 310 CMR 33.00

DPH Residential repair or removal of asbestos must be done in accordance with the State Sanitary Code. Notice of plans for abatement and removal must be submitted to the Local Board of Health.
Regulations: 105 CMR 410.353 (A)-(G) (Sanitary Code), 105 CMR 670.000 (RTK)

DLI DLI regulates worker protection in state, county and municipal government buildings. Asbestos abatement contractors, workers, and consultants must be licensed or certified by the Department of Labor and Industries. The Division of Industrial Safety enforces DLI's regulation. The Division of Occupational Hygiene is the governor's designee to review abatement plans of schools required by AHERA and will survey state, county and municipal buildings for asbestos. RTK requires training, labelling and recordkeeping.
Regulations: 453 CMR 6.00, 454 CMR 21.00 (RTK)

DCPO DCPO has contract standards for asbestos removal and repair in state-owned buildings.

(Adopted from "Department of Environmental Quality Engineering, Division of Air Quality Control, Asbestos Information and Resource Guide," November 1988.)

DEQE Division of Air Quality Control

DEQE has been delegated authority to enforce EPA's asbestos NESHAP regulation (National Emission Standards for Hazardous Air Pollutants). In addition to this authority, DEQE promulgated its own asbestos regulation on December 11, 1987. This regulation requires 20-day prior notification and strict work practices for all size asbestos abatement jobs. The goal of the regulation is to protect public health by preventing the release of asbestos emissions to the ambient air during building construction, renovation or demolition. DEQE ensures compliance with the asbestos regulation by inspecting abatement jobs and initiating enforcement actions through its four regional offices.

DLI Division of Occupational Hygiene

The Massachusetts Department of Labor and Industries (DLI) promulgated regulations for the removal, containment, or encapsulation of asbestos pursuant to its authority under M.G.L. c. 149. (118) These regulations contain two major components - work practice standards for asbestos abatement projects with notification to applicable state and local authorities (effective October 30, 1987) and licensing and certification requirements for asbestos contractors, workers, consultants and testing laboratories (effective May 2, 1988)

The regulations also establish three categories of asbestos abatement projects - Spot Repair, Asbestos Associated Projects and Asbestos Abatement Projects. Spot Repair projects involve less than three (3) linear or square feet of asbestos-containing material (ACM), with notification ten (10) days in advance of Spot Repair projects to DLI.

Asbestos Associated Projects involve work performed for purposes other than asbestos abatement but which may disturb asbestos or otherwise result in worker exposure to asbestos. Asbestos Abatement Projects consist of removal, enclosure, encapsulation, renovation, repair, demolition or any other work directly affecting (ACM).

Certification is required of any worker on asbestos projects greater than three (3) linear or square feet; licensure is required by any firm or contractor performing an asbestos abatement project. Training for certification is provided by state-certified educational entities and differs in substance and length depending upon the level and type of certification being sought.

The certification program embodies the standards established by the regulations for ensuring competence of entities performing the removal, enclosure or encapsulation of asbestos or ACM; performing asbestos surveys, sampling or hazard evaluation; preparing asbestos abatement plans or abatement contract specifications; monitoring asbestos abatement operations, and providing asbestos analytical work.

The asbestos regulations are applicable to all work, construction, demolition, alteration, repair and maintenance in any facility where such work involves asbestos or asbestos-containing material. The term facility includes not only schools and other public buildings, but also commercial, industrial and even single-family homes. In efforts to comply with these regulations, homeowners may request a Department of Public Health (DPH) survey to verify the presence of asbestos before implementing an asbestos abatement project. DLI also maintains a computer listing of certified asbestos abatement workers, available to homeowners or any other entity requesting this information.

REGULATION OF ASBESTOS IN OTHER STATES

Forty states have enacted some form of legislation pertaining to asbestos and which are summarized in Table 5-3. Many require licensing and certification of asbestos abatement workers similar to Massachusetts' regulations. Twelve states implemented licensing requirements in 1987, including Massachusetts.

The use of asbestos-containing materials in new buildings is regulated in some states. For example, California's asbestos regulations contain provisions which severely restrict sale, transfer, purchase, or manufacture of asbestos-containing products. Connecticut regulations prohibit the introduction into commerce of any asbestos-containing materials that may be used for construction without labeling that indicates the product contains asbestos and that asbestos may cause cancer when inhaled. Florida goes so far as prohibiting all use of asbestos or asbestos-based fiber materials in any buildings constructed after September 1983 that are financed with public funds or constructed for the express purpose of being leased by a government entity. (113)

Illinois established an Asbestos Abatement Authority in October 1987, charged with developing and implementing a program for identification and abatement of asbestos hazards in all state government buildings. The Illinois Attorney General's Office also has a specific Asbestos Litigation Division to investigate and initiate legal actions necessary to ensure maximum recovery of asbestos abatement costs for all state entities. (113)

Some states such as Nevada, North Dakota and Rhode Island set specific deadlines for filing claims for compensation based on asbestos-related diseases. A total of twelve states have statutes addressing the legal consequences of asbestos use, application, and manufacture, or liability that may occur because of asbestos abatement work. (113)

The City of Portland, Maine's Housing Code requires that ACM must be kept in good repair in all dwelling units that contain one or more rooms intended for residential use. (113)

TABLE 5-3

Summary of State Asbestos Laws

	Alaska	Ariz.	Ark.	Calif.	Colo.	Conn.	Del.	Fla.	Ga.	Hlaw.	Ill.	Iowa	Kan.	Ky.	La.
Scope Of Law															
Public Schools	X		X	X	X	X	X	X	X		X	X	X		X
Private Schools	X		X	X	X	X	X		X		X	X	X		X
State Government Buildings	X		X	X	X	X	X	X	X		X	X	X	X	X
Some Privately-owned Buildings	X		X	X	X	X	X	X	X			X	X		X
Administration															
Single Department			X								X	X	X	X	
Multi-agency or Cooperative	X	X		X	X	X	X	X	X	X					X
New Agency, Board, or Commission				X				X	X		X				X
Enforcement															
Cease and Desist/Injunctions					X			X	X	X			X		
Contractor/Worker Penalties	X		X	X	X	X	X	X		X	X	X	X		
Whistleblower Protection															
Owner Penalties				X											
Financing															
Bond Issues															
Special Funds			X	X							X			X	
Contractor and Worker Standards															
Licensing/Certification Required	X		X	X	X	X	X	X	X	X	X	X	X		X
Application Specifications				X	X	X						X	X		
Liability Insurance Proof			X					X			X				
Training Requirements	X		X	X	X	X	X	X	X	X		X	X		
License/Certification Examination	X		X	X	X			X		X					
Physical Examination							X			X		X	X		
Reciprocity with Other States						X	X		X						
License/Certificate on Worksite															
Required Work Records											X	X	X		
Worksite Inspections						X	X			X		X	X		
Inspection Standards															
Inspection for Asbestos Hazard	X			X	X	X	X	X	X		X			X	X
with Some Standards Specified				X				X			X				X
Notification of Results	X				X	X		X			X			X	
Records of Inspections	X			X			X	X			X			X	
Abatement Procedures															
Provisions for Abatement Procedure	X		X	X	X	X	X	X	X	X	X	X	X		X
with Some Standards Specified				X		X		X	X		X		X		X
Consumer Information Provisions					X										
Studies				X	X	X									

TABLE 5-3 (cont'd)

	Maine	Md.	Mass.	Mich.	Minn.	Miss.	Mo.	Neb.	N.H.	N.J.	N.Y.	N.D.	Ohio	Okla.	Ore.
Scope Of Law															
Public Schools	X	X	X	X	X	X	X	X	X	X	X	X	X	X	X
Private Schools	X	X		X	X		X	X	X	X	X		X	X	X
State Government Buildings	X	X	X	X	X		X	X	X	X	X	X	X	X	X
Some Privately-owned Buildings	X	X		X	X		X	X	X	X	X	X	X	X	X
Administration															
Single Department								X				X			
Multi-agency or Cooperative	X	X		X	X	X	X		X	X	X		X	X	X
New Agency, Board, or Commission			X	X							X				X
Enforcement															
Cease and Desist/Injunctions					X			X	X	X	X			X	
Contractor/Worker Penalties	X	X		X	X		X	X	X	X	X		X	X	X
Whistleblower Protection					X				X	X					
Owner Penalties															
Financing															
Bond Issues	X		X									X			
Special Funds					X	X		X					X		
Contractor and Worker Standards															
Licensing/Certification Required	X	X		X	X		X	X	X	X	X	X	X	X	X
Application Specifications		X						X					X		
Liability Insurance Proof				X											
Training Requirements	X	X		X	X		X	X	X	X	X	X	X	X	X
License/Certification Examination	X			X	X				X	X		X	X	X	
Physical Examination								X							
Reciprocity with Other States	X								X						X
License/Certificate on Worksite	X				X				X	X	X				
Required Work Records		X		X		X		X			X		X		
Worksite Inspections		X			X		X	X	X	X	X		X	X	X
Inspection Standards															
Inspection for Asbestos Hazard	X	X			X	X	X	X	X	X	X	X	X	X	
with Some Standards Specified		X				X	X	X	X		X		X	X	
Notification of Results						X	X	X	X						
Records of Inspections	X	X					X				X				
Abatement Procedures															
Provisions for Abatement Procedure	X	X	X	X	X	X	X		X	X	X	X	X	X	X
with Some Standards Specified	X	X		X	X	X	X		X	X	X		X		
Consumer Information Provisions									X				X		
Studies		X	X						X				X		

TABLE 5-3 (cont'd)

	R.I.	Tenn.	Tex.	Utah	Va.	Vt.	Wash.	Wis.
Scope Of Law								
Public Schools	X	X	X	X	X	X	X	X
Private Schools	X					X	X	X
State Government Buildings	X		X	X	X	X	X	X
Some Privately-owned Buildings	X		X	X		X	X	X
Administration								
Single Department				X		X	X	
Multi-agency or Cooperative	X				X			
New Agency, Board, or Commission			X					
Enforcement								
Cease and Desist/Injunctions			X					X
Contractor/Worker Penalties	X		X			X	X	X
Whistleblower Protection	X							
Owner Penalties	X							
Financing								
Bond Issues	X							
Special Funds	X							
Contractor and Worker Standards								
Licensing/Certification Required	X		X	X		X	X	X
Application Specifications	X		X					
Liability Insurance Proof								
Training Requirements	X		X				X	X
License/Certification Examination								X
Physical Examination								
Reciprocity with Other States								
License/Certificate on Worksite	X							X
Required Work Records			X					
Worksite Inspections	X		X					X
Inspection Standards								
Inspection for Asbestos Hazard	X			X	X			
with Some Standards Specified	X							
Notification of Results	X				X			
Records of Inspections	X							
Abatement Procedures								
Provisions for Abatement Procedure	X		X	X	X	X	X	
with Some Standards Specified	X				X	X	X	
Consumer Information Provisions	X							
Studies		X						

Note: This table does not reflect the extensive 1987 asbestos regulations promulgated in Massachusetts, entitled "The Removal, Containment, or Encapsulation of Asbestos."

The New York State Attorney General took a bold step in 1986 to remedy asbestos hazards in residences, by promulgating regulations which required owners of cooperative apartments or condominiums to inform prospective buyers if an asbestos hazard exists in the subject apartment or condominium. Under the regulation, the owner must also carry out an asbestos abatement plan. This second portion of the regulation was challenged and overturned by the State Appellate Court. The Court ruled that while the Attorney General may require asbestos hazard notification, removal of an asbestos hazard may not be mandated by the Attorney General. (98)

New York City has promulgated its own asbestos regulations. Of note is the requirement that a city-certified inspector must determine that no asbestos fibers will be released during any proposed work before such work can begin, including construction or renovation of a property. (98)

LOCAL RESPONSIBILITIES

Massachusetts law provides for broad Board of Health authority to protect the public, health, safety and welfare, to abate nuisances and to determine where noxious trades or waste storage, transport, or disposal may occur. Through delegation of the State Sanitary Code and authority from DAQC, local Boards of Health regulate residential asbestos conditions, outdoor dust pollution, indoor air conditions, and approve disposal of asbestos in locally owned landfills. Boards of Health may also establish reasonable regulations relating to asbestos to protect the public health pursuant to M.G.L. c. 111, Sec. 31, in so far as such regulations do not conflict with state and federal regulatory requirements.

FEDERAL STUDIES ON ASBESTOS

In February 1988 EPA completed a study mandated by AHERA, investigating the presence of asbestos hazards in public and commercial buildings throughout the United States. (111) Federal buildings, offices, factories, stores, hospitals and large apartment buildings were examined. Apartment houses with less than ten units and single-family homes were not considered. The purpose of this study was to help EPA determine whether federal regulation of such public and commercial buildings is necessary.

Of 3.6 million public and commercial buildings in the United States, 733,000 contain friable asbestos. EPA found that 501,000 buildings contained damaged friable asbestos and 317,000 were plagued with significantly damaged asbestos as defined by AHERA. In this context, AHERA defines the term "damage" as meaning "circumstances in which friable asbestos-containing material or its covering is damaged, deteriorated, or delaminated" and "significant damage" as meaning "circumstances in which friable asbestos-

containing material or its covering is significantly damaged, deteriorated, or delaminated."

Given the results of this study, EPA recommended that no regulatory action be taken to abate these conditions in public and commercial buildings for at least three years. EPA concluded that before embarking on a comprehensive and expensive national asbestos abatement program for commercial and public buildings, the AHERA school program ought to be completed and evaluated. Once the school program is completed, EPA contends, legislators will be better able to determine if comprehensive inspection and regulation of public buildings is necessary.

EPA suggested accelerating the accreditation program for asbestos abatement workers, preparing new guidelines for asbestos thermal insulation, ensuring better enforcement of existing asbestos-related rules, and assessing the on-going effort to abate asbestos hazards in schools.

In response to several directives, especially from the Asbestos School Hazard Abatement Act (ASHAA) of 1985, and AHERA of 1986, EPA conducted asbestos air monitoring studies in public buildings. EPA released its report in May 1988. The air monitoring studies were intended to provide data on existing asbestos exposure levels inside buildings with asbestos-containing material as well as ambient outdoor levels. (119) The study compared asbestos air levels in buildings with no asbestos (Category 1), asbestos in good condition (Category 2), and asbestos with some significant damage or with numerous areas of moderate damage (Category 3).

Differences in airborne levels of asbestos were found to be small in absolute magnitude. However, a general trend was detected, with average airborne asbestos levels higher in buildings with asbestos-containing material than in buildings without asbestos-containing material.

The Consumer Product Safety Commission completed a study of asbestos levels in 45 asbestos-containing homes in Cleveland, San Francisco and Philadelphia. No indoor asbestos background levels were found to be any greater than outdoor levels. (120)

No measurements exceeded EPA's recommended post-abatement level of 0.01 fibers per cubic centimeter for fibers greater than five microns in length. Air was monitored in two places inside each home and at one location outside each home. Rooms containing the asbestos product and rooms most heavily used by the occupants were among the locations tested. (120)

The CPSC study represents a week-long "snapshot" of air levels of asbestos fibers. The authors therefore caution against the extrapolation of this data for the purpose of assessing asbestos-related risk in homes throughout the United States. (120)

FEDERAL REGULATION OF ASBESTOS

EPA and OSHA share most of the burden of regulating the use of asbestos in both occupational and non-occupational settings. Regulations to date focus on controlling occupational exposure to asbestos and abatement of asbestos hazards in schools. These federal laws and regulations dealing with asbestos are summarized below.

OSHA (U.S. Dept. of Labor, Occupational Safety and Health Administration)

"General Industry Standard" for Occupational Exposure to Asbestos
Title 29 CFR Part 1910.1001

Establishes standards to protect workers in occupations where they are exposed to airborne concentrations of asbestos fibers in all industries, excluding construction work.

"Construction Standard" for Occupational Exposure to Asbestos
Title 29 CFR Part 1926.58

Establishes standards to protect workers in construction work occupations where they are exposed to airborne concentrations of asbestos fibers.

EPA (U.S. Evironmental Protection Agency)

"NESHAPs" (National Emission Standards for Hazardous Air Pollutants)
Establishes standards for demolition and renovation projects and procedures for asbestos emission control.

"Worker Protection"
Title 40 CFR Part 763,

Final ruling implementing OSHA's workplace standards for construction work for asbestos abatement projects conducted by state and local government employees.

"AHERA" (Asbestos Hazard Emergency Response Act of 1986)
Title 40 CFR Part 763

Details how local education agencies (LEA) must inspect, sample, assess and develop management plans for all asbestos-containing building material; creates an accreditation plan for persons who inspect for asbestos, develop management plans, or design/conduct response actions must complete an EPA-approved training course and pass an exam to become certified. States can adopt this plan or develop their own at least as stringent.

"SDWA" (Safe Drinking Water Act)

42 U.S.C. 300(f) et seq.

Requires annual testing of municipal water supplies to detect concentration levels of asbestos.

"FWPCA" (Federal Water Pollution Control Act or Clean Water Act)
Establishes standards for facilities which discharge asbestos into public sewers or navigable waters.

CPSC (Consumer Product Safety Commission)
"Consumer Product Safety Act" and "Hazardous Substances Act"
Provide basis for CPSC authority to establish standards and to institute recalls or bans in order to address identified hazards.

DOT (U.S. Department of Transportation)
"Hazardous Materials Transportation Act"
Regulates the conditions under which asbestos may be transported.

History of Asbestos Regulation

Table 5-4 presents an historic perspective of federal regulation of asbestos from 1971 through the present. EPA first considered banning the use of asbestos altogether in its 1986 proposed Asbestos Ban and Phase Down Rule, which if ever finalized, will phase out the commercial use of asbestos over the next ten years. The first asbestos products that would be banned by this rule include roofing felt, vinyl-asbestos floor tile, clothing, flooring felts and asbestos cement pipes. (121) No progress on this rule has occurred to date.

AHERA

AHERA provides for the establishment of federal regulations which require the inspection of ACM and implementation of appropriate response actions (Management Plans) in schools with periodic reinspection of schools following such response actions. AHERA mandated the EPA study completed in February of this year, surveying the extent of danger to human health posed by asbestos in public and commercial buildings throughout the Commonwealth. It is important to note that AHERA does not establish safe exposure standards for asbestos; appropriate abatement action is dictated simply by the presence of asbestos and the extent to which it is friable and posing a risk to human health.

TABLE 5-4

MAJOR FEDERAL ACTIONS ON ASBESTOS: A CHRONOLOGY

Compiled by the Institute for Environmental Education

Occupational Standards

OSHA	June 1972. "Permanent standard" for occupational exposure of 5 f/cc, to be lowered to 2 f/cc in 1976
OSHA	October 1975. Proposed lowering standard to 0.5 f/cc.
OSHA	July 1976. 2 f/cc standard became effective.
NIOSH	December 1976. Recommended that OSHA lower the standard to 0.1 f/cc.
MSHA	March 1976. 2 f/cc standard in coal mines.
MSHA	November 1978. 2 f/cc standard in metal and nonmetallic mines (includes sand, gravel, and crushed stone operations).
OSHA	November 1983. Issued emergency temporary standard (ETS) of 0.5 f/cc.
OSHA	November 1983. ETS stayed pending legal arguments by asbestos industry.
OSHA	March 1984. ETS overturned in Federal District Court.
OSHA	June 20, 1986. (29 CFR 1910 and 1926). OSHA issued a revised general industry standard and construction standard. Both standards establish an action level of 0.1 f/cc as an eight hour TWA and a permissible exposure limit of 0.2 f/cc as an eight hour TWA.

Air Emissions

EPA/NESHAP	March 1971. Asbestos listed as a hazardous air pollutant.
EPA/NESHAP	April 1973. 40 CFR Part 61, subparts A & B (38 FR 8826). National Emission Standards for Hazardous Air Pollutants Asbestos Standard. No visible emissions standard for milling and manufacturing of asbestos products and

TABLE 5-4 (cont'd)

	demolition of buildings. Prohibited spray application for most uses of friable materials containing more than 1% asbestos.
EPA/NESHAP	October 1975. Waste collection and disposal included under the no visible emissions standard. Added several processing industries to those already covered.
EPA/NESHAP	June 1978. Extended prohibition to cover all uses of friable spray-on material and no visible emissions standard to cover all friable asbestos-containing materials during demolition.
U.S. Supreme Court	January 1978. Decision in the Adamo Wrecking Company case ruled that EPA did not, prior to the 1977 Clean Air Act amendments, have the authority to enforce work practice requirements as emission limits thus invalidating those parts of the NESHAP asbestos standard.
U.S. Congress	1978. Clean Air Act amended. Work practice procedures now can be used as emission standards.
EPA/NESHAP	April 5, 1984 (49 FR 13661). NESHAP asbestos standard repromulgated. Reinstatement of the work practice provisions originally deemed unenforceable in January 1978 Adamo Wrecking Company case.
	Asbestos in Schools
EPA	March 1979. Through the OTS, EPA initiated a technical assistance program to help schools identify and control friable asbestos-containing materials.
EPA/TSCA	September 1979. ANPR on asbestos-containing materials in schools.
EPA/TSCA	September 1980. Proposed rule on identification and notification of friable asbestos-containing materials in schools.
Education	September 1980. Under the Asbestos School Hazard Detection and Control Act, the Department of Education proposed a rule to establish a grant and loan program to reimburse schools for detecting and controlling friable asbestos-containing materials.
Education	January 1981. Final rule. Funds have not been appropriated to conduct this program.

TABLE 5-4 (cont'd)

EPA/TSCA	May 27, 1982 (40 CFR Part 763). Friable Asbestos-Containing Materials in Schools; Identification and Notification Rule. Final rule was published on identification and notification of friable asbestos-containing materials in schools.
EPA/TSCA	February 1983. EPA granted in substantial part a Section 21 petition from the Service Employees International Union to commence regulatory action on asbestos abatement in schools and buildings.
EPA/TSCA	August 10, 1984. Asbestos School Hazard Abatement Act (ASHAA) was signed into law. EPA provided assistance in the form of loans and/or grants to schools for the abatement of serious asbestos hazards. This regulation authorized $600 million in grant and loan money over a seven year period.
EPA/TSCA	July 12, 1985 (50 FR 28530). Asbestos Abatement Projects; Worker Protection; or the "Worker Protection Rule" was promulgated. This regulation was promulgated to protect state and local public employees involved in asbestos abatement who were not covered by asbestos standards issued under the state plans approved by OSHA. This was an immediately effective proposed rule.
EPA/TSCA	April 25, 1986 (51 FR 15722). Asbestos Abatement Projects; Worker Protection; Final Rule was published. In this rule, EPA announces that it will reissue a revised rule to reflect OSHA revisions in EPA's regulations.
EPA/TSCA	February 25, 1987 (52 FR 5618). Asbestos Abatement Projects; Worker Protection; Final Rule. EPA incorporates changes in this rule that make it comparable to OSHA general industry and construction standards. Additionally, EPA institutes new requirements for engineering and work practice controls, worker training requirements and EPA notification of asbestos abatement projects.
EPA/TSCA	October 22, 1986. Asbestos Hazard Emergency Response Act (AHERA) was signed into law. The Act required EPA to promulgate rules regarding inspection of all public and private school buildings, identification of circumstances requiring response actions, description of appropriate response actions, implementation of response actions, establishment of reinspection and periodic surveillance program, establishment of operations and maintenance program, preparation of management plans and transportation and disposal of waste ACM.

TABLE 5-4 (Cont'd)

EPA/TSCA	April 30, 1987 (52 FR 15820). Asbestos-Containing Materials in Schools; Proposed Rule and Model Accreditation Plan; Rule was published. This regulation addressed the requirements of AHERA as stated above. Model Accreditation Plan outlines requirements for persons who inspect for asbestos, develop management plans and design or conduct response actions. The Plan was put into effect upon publication.
EPA/TSCA	October 30, 1987 (40 CFR Part 763). Asbestos-Containing Materials in Schools; Final Rule and Notice was published.
	Commercial Use of Asbestos
CPSC	December 1977. Rules prohibiting use of asbestos in consumer patching compounds and emberizing agents.
EPA/TSCA	October 1979. ANPR with CPSC announcing intent to consider regulations of commercial uses of asbestos.
EPA/TSCA	December 1979. ANPR modification.
EPA/TSCA	September 1980. Proposed rule under section 8(a) to require reporting of production and exposure data on asbestos.
EPA/TSCA	July 1982. Final rule under section 8(a) to require reporting of production and exposure data on asbestos.
	Water Emissions
EPA/FWPCA	February 1974. Effluent guidelines for asbestos manufacturing point sources and new source performance standards.
	Water Disposal
EPA/RCRA	May 1980. Asbestos listed as a hazardous waste in proposed rules.
EPA/RCRA	November 1980. When issuing interim final rules on portions of the disposal regulations, EPA stated that they would "temporarily defer" promulgation of the listing of asbestos while investigating the extent to which NESHAP facilities afford comparable protection.

TABLE 5-4 (Cont'd)

EPA/NESHAP	April 1984. Disposal of asbestos waste is regulated by the NESHAP asbestos standard.
	Other Actions
DOT	August 1979. Rule to require controls during transportation of friable asbestos.
DOT	November 21, 1986. Regulations promulgated pursuant to the disposal of asbestos wastes.
FDA	March 1975. Rule to prevent release of asbestos from filter used for some drugs.
FDA	January 1976. Rule to revoke permission to use the electrolytic diaphragm process for salt.

Under AHERA and its regulations, Local Education Authorities (LEAs) must complete the following tasks, some within specific timeframes as noted:

(1) Inspection of building(s) by accredited persons and laboratories for both friable and nonfriable ACM;

(2) Creation of a Management Plan to develop response action to asbestos hazards. In these Management Plans, LEAs possess several options in abating asbestos hazards - develop operation and maintenance plans for ACM not posing a public health hazard or repair, encapsulate, enclose or remove asbestos. The Management Plans were due by October 12, 1988, although LEA's had the opportunity to defer submittal of their Management Plans until May 1989;

(3) Implement the Management Plans in its buildings by July 1989 and complete them in a "timely fashion;"

(4) Conduct periodic surveillance and reinspection;

(5) Assure proper training and awareness (EPA funded training programs are available); and

(6) Maintain records of asbestos monitoring and surveillance activities.

OSHA

OSHA adapted two regulatory standards for occupational exposure to asbestos, one for general industry and one for construction sites. These standards include an action level, a permissible exposure level and a short term exposure level.

The action level is set at 0.1 fibers per cubic centimeter calculated as an 8-hour time-weighted average. Initial air monitoring for asbestos is required where employees are or may reasonably be expected to be exposed to airborne concentrations of asbestos at or above this action level. Employees must be given the opportunity to observe monitoring activities and be notified of monitoring results. Monitoring records must be retained for thirty (30) years. Medical surveillance of employees is also required at the action level as well as extensive employee information and training. (122)

OSHA's permissible exposure level (PEL) for asbestos was reduced ten-fold in 1986 to 0.2 fibers/cubic centimeter calculated as an 8-hour time-weighted average. The employer must ensure that no employee is exposed to an airborne concentration of asbestos fibers in excess of this PEL for either industrial or construction site workers.

Where the PEL is exceeded, regulated areas must be established to which access is limited, warning signs are posted and protective clothing and respiratory protection are provided. Engineering controls of the source(s) of asbestos must be in place and work practice controls established. All

engineering controls must be employed before considering work practice controls since such controls are subject to worker behavior and thus worker error. Hygiene facilities and protective clothing must be available as well as a written compliance program. (122)

Recently, OSHA established a short term exposure limit for asbestos, set at 1 fiber/cubic centimeter over a sampling period of thirty (30) minutes. (123)

MEASUREMENT AND MONITORING OF ASBESTOS

Neither a regulatory safe exposure level nor one born of scientific consensus exists for asbestos. Still, certain concentrations of asbestos fibers in air trigger monitoring requirements and sometimes abatement activities. In monitoring for asbestos hazards, four types of microscopic analysis techniques are used and must be carried out only by federally acredited laboratories.

Polarized Light Microscopy

Polarized Light Microscopy (PLM) is used for the analysis of bulk samples and building materials for asbestos content under the "Interim Method for the Determination of Asbestos in Bulk Insulation Samples." For inspection and sampling purposes, suspected ACM must be inspected visually, touched to determine friability, then homogeneous areas of friable and non friable ACM must be identified. Among those homogeneous areas not already assumed to contain asbestos, bulk samples must be obtained. (124)

PLM allows identification of asbestos by viewing the morphology of fibers using stereoscopic polarized light examination. A visual estimate of the percent of each fiber type present is made. Fibers less than twenty (20) microns in length are not readily determined and fibers less than one (1) micron in width are not even discernible. (125)

Phase Contrast Microscopy

Phase contrast microscopy (PCM) is widely used for assessing worker exposure to airborne asbestos fibers using NIOSH 7400 Method and the OSHA Reference Method. Fibers less than .25 microns in width cannot be detected. PCM also does not distinguish between asbestos fibers and all other types of fibers. (125) This ability to differentiate asbestos fiber types will be especially important in the future as the variability in toxicity among fiber types is better understood.

Transmission Electron Microscopy/Scanning Electron Microscopy

Transmission Electron Microscopy (TEM) embodies state-of-the-art technology for analyzing airborne concentrations of asbestos fibers. Fibers as small as 0.1 micron in length are discernible. For this reason, the AHERA rule requires the use of TEM for measuring airborne asbestos fiber concentration for clearance of post removal abatement sites.

However, until October 1989, PCM may be used for clearance of asbestos abatement projects less than or equal to 3000 square feet or 1000 linear feet. Between October 1989 and October 1990, PLM may be used for clearance only for abatement projects less than 1500 square feet or 500 linear feet. (124)

Asbestos fiber types can also be positively identified through the use of TEM analysis. Scanning Electron Microscopy provides similar benefits, but no established analytical protocol has been established for this type of microscopy.

ASBESTOS HAZARD MITIGATION

Alternatives to Asbestos

The physical and chemical properties of asbestos resulted in its ubiquitous use in building materials and for fire protection. Now that exposure to those once valued properties has been correlated with lung cancer, mesothelioma and asbestosis, alternatives to asbestos have been explored.

Several asbestos substitutes already have been identified and used in place of asbestos in a number of products. These substitutes include flexible graphite, carbon and graphite yarn, glass fibers, aramid, polybenzimidazole (PBI), polytetrafluoroethylene (PTFE), ceramics and mica. (126) No single material matches up to asbestos with its useful array of properties - high tensile strength, flexibility, durability, resistance to fire, heat and corrosion, and friction properties.

Flexible graphite has proven to be a credible asbestos substitute for packing, withstanding even higher temperatures than asbestos. For gaskets, chlorite/graphite sheet has been used extensively. Calcium silicate serves as an asbestos substitute in plant and equipment insulation. (126) Rather than search for an asbestos substitute, Ceram-Sno, Inc. of Quebec has researched means to make asbestos safe by developing a process that modifies the surface of chrysotile asbestos fibers. (99)

Current public health problems associated with asbestos, as well as related lawsuits and insurance coverage difficulties may drive markets using asbestos towards these and other substitute materials. Not only must asbestos

replacements function properly and be cost effective, but they must not represent a similar or worse health threat than asbestos. Some evidence exists that manmade mineral fibers, specifically rockwool and glass fibers, have the ability to damage DNA. (127) Careful consideration must be given to the potentially toxic and carcinogenic effects of each such alternative to avoid the extensive toll asbestos has taken on human health.

Asbestos Removal Versus Enclosure or Encapsulation

Most experts agree that asbestos poses no danger if it is in good condition and undisturbed. For this reason, federal and state requirements provide latitude for building owners to decide whether wholesale removal of asbestos is warranted or mitigation of potential asbestos hazards through enclosure or encapsulation. Total removal, however, is the only way to eliminate any potential asbestos health hazard since asbestos may not remain undisturbed. Pipe leaks, fires, aging of asbestos-containing materials and remodeling work may all cause asbestos fibers to become airborne.

The encapsulation technique coats a binding or sealing agent onto asbestos or ACM to prevent the release of asbestos fibers. Enclosure means generally covering or wrapping of ACM in, under or behind air-tight barriers. (118)

Encapsulation and enclosure do not eliminate the potential for liability. For this reason, banks and other financial institutions may be reluctant to finance building renovations that might disturb asbestos or ACM. A trend toward lenders requiring asbestos clean-up before mortgages on commercial buildings will be approved may be occuring. (128)

As long as asbestos is present, even if enclosed or encapsulated, long-term costs will be incurred for periodic reinspection, maintenance, employee education and training concerning the health threats posed by asbestos, record-keeping costs and purchase of glove bags, HEPA filter-equipped vacuums or other equipment for dealing with emergency situations.

Complete removal of asbestos alleviates the long-term problems of liability and maintenance, but may result in short-term worker or even public exposure to asbestos fibers if the abatement work is not implemented properly. Removal costs vary relative to five variables - labor costs, type of asbestos being removed, phasing of the abatement work, the type of asbestos application and the costs of replacement materials (129).

Liability Insurance

In Massachusetts, insurance companies have refused long-term worker compensation and liability insurance to asbestos abatement contractors. (117) Several reasons may be set forth for this dilemma. First, no safe exposure level exists for asbestos - either through regulation or scientific consensus.

Second, asbestos is a recognized carcinogen. Third, only recently have uniform work practice standards been established in Massachusetts.

The passage of Massachusetts regulations establishing asbestos work standards as well as licensing and certification requirements represents a critical step in alleviating the problem of obtaining liability insurance by creating standards for what constitutes proper asbestos abatement practices. The regulations "shall establish and/or constitute...minimum standards used by insurers, on or after May 2, 1988, in the inspection of risk, measurement of hazards and the determination of the adequate and reasonable rate or rates of insurance..." (118)

A fourth reason for the difficulty in obtaining liability insurance is the present lack of judicial uniformity with regard to the proper theory of asbestos liability. (130) Four different theories of asbestos insurer liability exist as interpreted by five U.S. Courts of Appeals - the exposure theory, manifestation theory, continous injury theory and injury-in-fact theory.

Under the exposure theory, insurers providing coverage upon the initial inhalation of asbestos fibers and upon each subsequent exposure are liable to indemnify the manufacturer. Under the manifestation theory, liability is not incurred until the victim's symptoms are clinically evident or the condition is reasonably capable of medical diagnosis. The continuous injury theory, on the other hand, holds that inhalation exposure, exposure in residence (disease development), and manifestation all trigger coverage. Finally,the injury-in-fact theory concludes that a determination as to when actual injury occurred triggers liability coverage. (130)

Resolution of this judicial uncertainty along with the passage of Massachusetts asbestos regulations may eliminate asbestos abatement contractor difficulties in obtaining affordable liability insurance.

Chapter 6: ENVIRONMENTAL TOBACCO SMOKE

ENVIRONMENTAL TOBACCO SMOKE DEFINED

An overwhelming amount of evidence establishes cigarette smoking as the single largest preventable cause of premature death and disability in the United States. (131) In Massachusetts alone, the Department of Public Health reports that cigarette smoking results in 8,500 deaths annually. (132) Since the smoke to which nonsmokers are exposed is qualitatively similar to tobacco smoke inhaled by a smoker, concern for the "involuntary smoker" has arisen.

Involuntary smokers are nonsmokers exposed to environmental tobacco smoke (ETS) in their home, work or social environments. ETS is comprised of a conglomerate of chemical substances, including aldehydes (such as formaldehyde and acrolein), aromatic hydrocarbons, carbon monoxide, nicotine, nitrogen oxides, nitrosamines, phenols, polynuclear aromatic hydrocarbons, and respirable particulate matter. Respirable particulates are less than 2.5 microns in diameter and can penetrate deeply into the lungs where significant health damage may occur.

ETS originates from sidestream smoke (SS) emanating from the burning portion of a cigarette, cigar or pipe; smoke diffusing through the cigarette or cigar paper; and mainstream smoke (MS) which is exhaled by the smoker. ETS differs from the MS inhaled by the smoker to some extent due to the dilution of ETS throughout a particular enclosed volume of space as well as the aging of ETS constituents. Aging involves such processes as coagulation, evaporation, and settling of ETS particles. (133)

MS and SS differ qualitatively since SS components are generated at a lower temperature of combustion, thus resulting in higher concentrations of organic constituents in SS than in MS, including some carcinogens. While a comparison of the chemical composition of smoke inhaled by smokers and ETS suggests that the health effects are qualitatively similar, SS has been shown to contain greater amounts of ammonia, benzene, carbon monoxide, nicotine, and the carcinogens 2-napthylamine, 4-aminobiphenyl, N-nitrosamine, benz(a)anthracene, benzene and benzo(a)pyrene per milligram of tobacco burned.

Therefore, SS appears to be more carcinogenic than MS. This fact must be balanced against the dilution and aging of ETS versus the MS which is directly inhaled by smokers. As a result, nonsmokers take in a much smaller dose of ETS than smokers take in of MS. Table 6-1 shows the lesser concentrations of toxins and carcinogens in ETS relative to the MS which is inhaled by smokers.

TABLE 6-1 Concentrations of toxic and carcinogenic agents in nonfilter cigarette mainstream smoke and in environmental tobacco smoke (ETS) in indoor environments

	Mainstream Smoke		Inhaled as ETS constituents during 1 hour			
			Range		Episodic high values[1]	
Agent	Weight	Concentration	Weight	Concentration	Weight	Concentration
Carbon monoxide	10–23 mg	24,9000–57,300 ppm	1.2–22 mg	1–18.5 ppm	37 mg	32 ppm
Nitrogen oxide	100–600 μg	230,000–1,400,000 ppb	7–90 μg	9–120 ppb	146 μg	195 ppb
Nitrogen dioxide	<5 μg	<7,600 ppb	24–87 μg	21–76 ppb	120 μg	105 ppb
Acrolein	60–100 μg	75,000–125,000 ppb	8–72 μg	6–50 ppb	110 μg	80 ppb
Acetone	100–250 μg	120,000–300,000 ppb	210–720 μg	150–500 ppb	3,500 μg	2,400 ppb
Benzene[2]	12–48 μg	11,000–43,000 ppb	12–190 μg	6–98 ppb	190 μg	98 ppb
N-Nitrosodimethylamine[3]	10–40 ng	9–38 ppb	6–140 ng	0.003–0.072 ppb	140 ng	0.072 ppb
N-Nitrosodiethylamine[3]	4–25 ng	3–17 ppb	<6–120 ng	<0.002–0.05 ppb	120 ng	0.05 ppb
Nicotine	1,000–2,500 μg	430,000–1,080,000 ppb	0.6–30 μg	0.15–7.5 ppb	300 μg	75 ppb
Benzo[a]pyrene[4]	20–40 ng	5–11 ppb	1.7–460 ng	0.0002–0.04 ppb	460 ng	0.04 ppb

NOTE: Values for inhaled mainstream smoke components were calculated from values in Table 2 and on a respiratory rate of 10 L per minute. Values for carbon monoxide and nicotine represent the range in mainstream smoke of U.S. nonfilter cigarettes as reported by the U.S. Federal Trade Commission (1985). Data under ETS are derived from Tables 8 through 15, with data from the unventilated interior compartments of automobiles excluded (Badre et al. 1978).

[1] The designation "episodic high values" was chosen to classify those data in the literature that require confirmation.

[2] Human carcinogen according to the IARC (Vainio et al. 1985) and suspected carcinogen according to the ACGIH (1985).

[3] Animal carcinogen according to the IARC (Vainio et al. 1985).

[4] Suspected human carcinogen, according to the IARC (Vainio et al. 1985) and according to the ACGIH (1985).

(U.S. Dept. of Human Health and Services, December 1986)

HEALTH EFFECTS OF EXPOSURE TO ETS

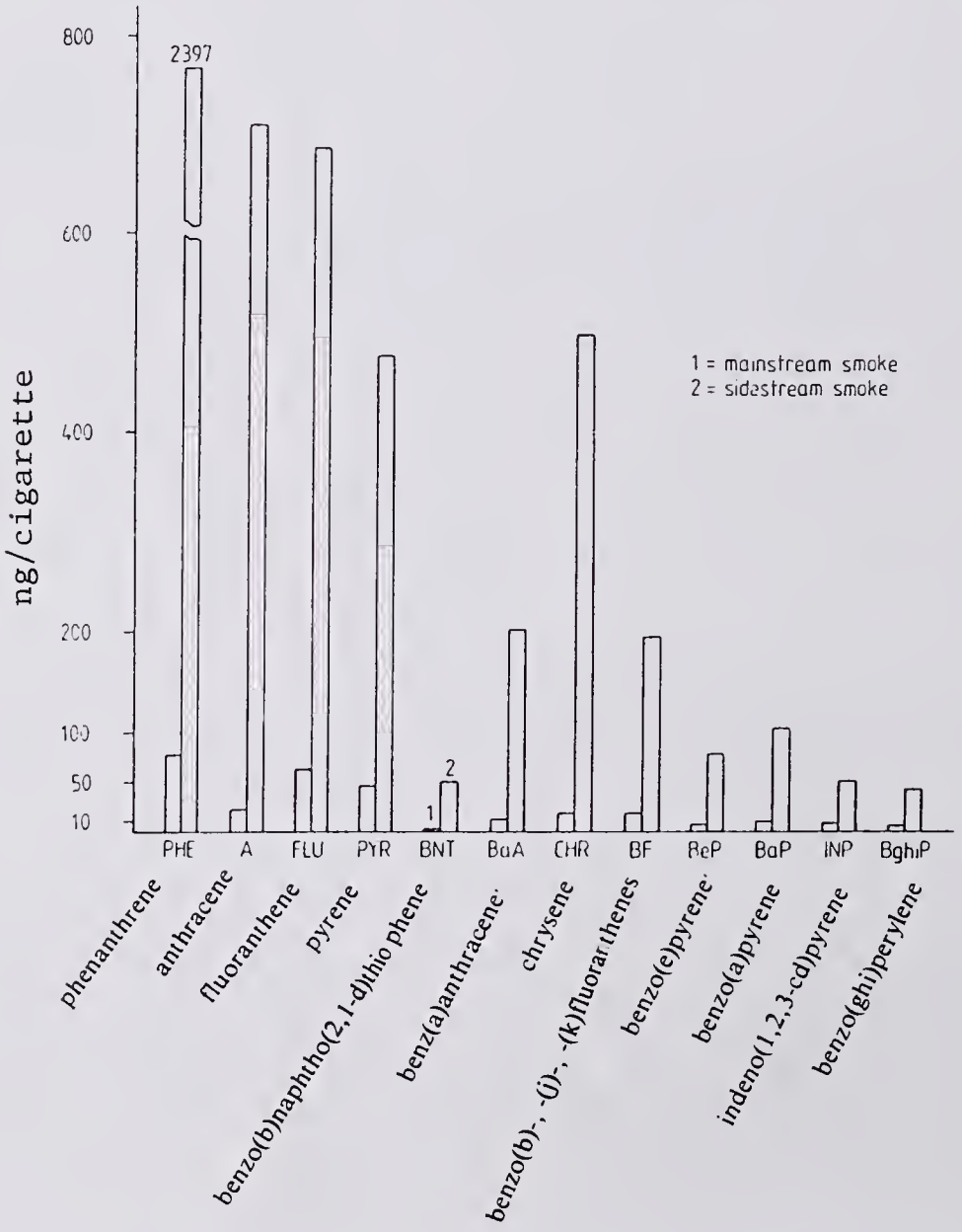
Smoke Constituents

Over 3800 compounds have been detected in cigarette smoke. (134) Some cancer-causing substances, or carcinogens, comprise the more hazardous of these constituents. Of the two primary types of cigarette smoke, sidestream smoke (SS) and mainstream smoke (MS), SS contains higher concentrations of a number of toxins. Recent evidence suggests that modern, filtered cigarettes produce even more toxic SS than previous, unfiltered designs. (135)

Differences in burning temperature account for most of the variability of waste products, particularly of the organic compounds. The tip of the cigarette, which generates SS, burns at approximately 350 degrees Centigrade, a significantly lower temperature than the more proximal portion, which produces MS. In contrast to MS, SS produces higher concentrations of nitrogen dioxides and amines, which in turn react and generate increased levels of N-nitrosamines. A number of N-nitrosamines are known or suspected carcinogens. Other toxic compounds at higher concentrations in SS than MS include ammonia, acrolein, carbon monoxide, nicotine, and some carcinogenic polycyclic aromatic hydrocarbons. See Figure 6-1 for a comparison between levels of some polycyclic aromatic hydrocarbons found in MS and SS. (136)

FIGURE 6-1

Ratio of Various PAH in Main and Sidestream Smoke



(Grimmer, Gernot, et al., 1987)

The toxicity of the primary constituent of ETS, SS, is undisputed. However, ETS is considerably more dilute than MS inhaled by smokers. The effects of long-term, low-level exposure to these toxins and carcinogens continue to be disputed. Several studies have shown small but significant increased cancer risks associated with ETS exposure. Moreover, exposure to other environmental pollutants, such as radon, may multiply the adverse effects of these pollutants or of ETS alone. Although studies of health effects are continuing, evidence to date suggests that exposure to tobacco smoke indeed imperils the health of passive smokers.

Acute Effects of ETS Exposure

Eye, nose and throat irritation are the most commonplace acute effects of tobacco smoke. Some of the major irritants in ETS are acrolein, toluene, ammonia, nitrogen oxides, sulfur dioxide, phenols, respirable particulates, and particles which can be inhaled. (131) The level of irritation seems to correspond well with the intensity and the length of smoke exposure. (137)

Respiratory Symptoms and Infections

Children

Cough, phlegm, and wheeze are three chronic respiratory symptoms which have been studied extensively. A U.S. study of five to nine year-olds found persistent wheeze significantly associated with parental smoking. (138) Two English studies, one of eight to nineteen year-olds, and one of twelve to thirteen year-olds found cough significantly associated with parental smoking, particularly maternal smoking. (139) Another U.S. study found no trend for increased cough, phlegm, or wheeze among children exposed to ETS. (140) Not all studies of involuntary exposure have corrected for such confounding factors as active smoking in children. The literature suggests a strong correlation between these health symptoms and passive smoking.

Several studies of respiratory diseases among children exposed to ETS seem to indicate increased incidence of respiratory illnesses, such as bronchitis and pneumonia. A six-city survey of American children between five and nine years old reported a significant association between maternal smoking and respiratory illnesses, specifically before age two and during the year prior to the study. When maternal respiratory symptoms were accounted for, the linear relationship of cigarette number and degree of illness was preserved. (141) See Figure 6-2 for the odds ratios of respiratory ilnesses and symptoms from this study. Another U.S. study of five to fourteen year-olds with smoking parents found significantly increased incidence both of chest illness before age two and chest illness during the year before the study. (143)

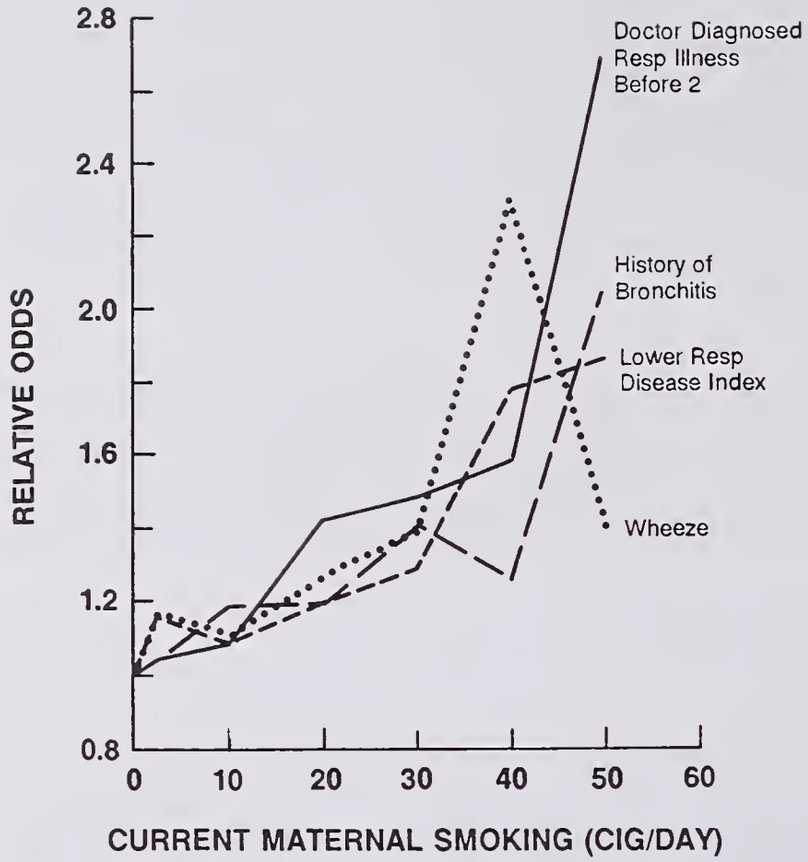


FIGURE 6-2 Relative odds of respiratory illness or symptoms versus average daily cigarette smoking by the child's mother. Reference value is zero cigarettes per day.

National Research Council, Environmental Tobacco Smoke-Measuring Exposures and Assessing Health Effects, 1986

Other studies have demonstrated increased risk for respiratory illnesses among children exposed to ETS. In 1986, the Surgeon General reviewed many such studies. He concluded that young children are particularly susceptible to health effects from ETS. (131)

Smoking mothers expose their unborn children to tobacco smoke constituents via the placenta, sometimes resulting in lower birth weights. (144) There may be other lasting consequences, as low birthweight is often an indicator of more severe effects on development. Some researchers have suggested that increased risk of illness may be due to in utero exposure to tobacco smoke, rather than environmental exposure after birth. (144)

Some studies have linked chronic ear infections and middle ear effusions in children with household exposure to ETS. ETS exposure may synergistically increase the risk of persistent middle ear effusions in children already suffering from nasal allergies and recurrent inflamation of the middle ear. (134)

Adults

As noted by the Surgeon General, adult exposure to environmental tobacco smoke may not be significantly associated with increased respiratory symptoms. At least two studies of spouses of smoking men found no relationship between cough, phlegm, or wheeze and exposure to ETS. (145)(146)

Asthma

Research on asthma suggests that exposure to ETS may aggravate asthma symptoms and may increase the severity of asthma. One study found that asthmatic children exposed to maternal smoke had nearly 50% more symptoms than asthmatic children not exposed to ETS. (147) Several studies found no increased incidence of asthma in children of smoking parents (140, 148), while another study attributed 18-23% of all childhood asthma to maternal smoking. (149)

Lung Function

The previous study noting increased asthma symptoms in children of smoking mothers also noted decreased scores in several lung function tests. They measured a 13% lower level of function in one pulmonary test, FEV_1. (147) FEV_1 is a standard lung function test. The acronym stands for forced expiratory volume, measured at the first second during expiration. FEV_1 varies with subject size and particularly with subject age. See Figure 6-3 for an average rate of growth of FEV_1 before age twenty, and an average rate of decline after age twenty. Figure 6-3 also indicates increased rates of decline (curve B) which can result from such factors as active smoking. FEV_1 would be expected to decline more rapidly with age from other lung diseases, as well.

Several studies have corroborated this finding of reduced expected levels of FEV_1 in children of smoking mothers, compared to children of nonsmoking mothers. In addition, some of these studies detected a reduced rate of growth in this measurement of lung function. (150)(151)

The Surgeon General has expressed great concern over the long-term effects of reduced growth in lung function from exposure to ETS. (131) It is not known if a reduced rate of growth of FEV_1 in children exposed to passive smoke will accelerate the normal rate of decline after age twenty. It is also possible these children never attain the levels reached by most young adults at approximately age twenty. Such impaired development is a serious risk factor that can predispose these children to other diseases later in life.

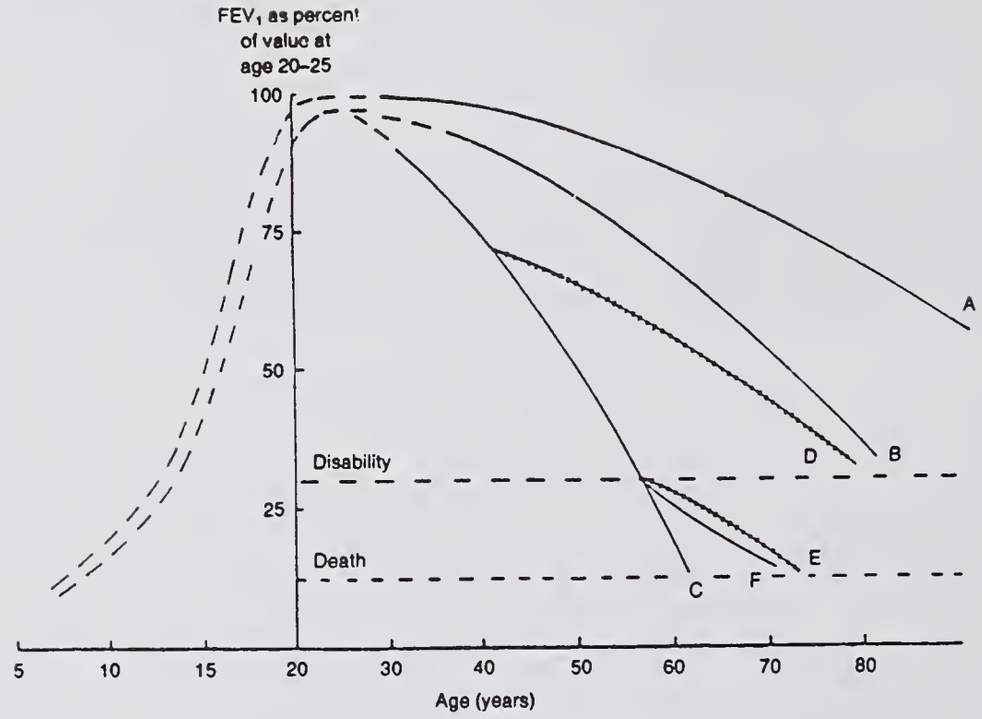


FIGURE 6-3 Theoretical curves representing varying rates of change in FEV_1 by age

NOTE: Curve A, normal decline in FEV_1 (forced expiratory volume in 1 second); curve B, accelerated decline in FEV_1 with cigarette smoking; curve D, the effect of smoking cessation, also seen in disabled individuals (curve E); disability-related decline often continues as a variable rate (curves C and F).

SOURCE: Speizer and Tager (1979).

Cardiovascular Disease

ETS may aggravate symptoms of preexisting coronary disease and two recent studies have linked it to heart disease mortality (152, 153). Another study found no correlation between ETS and ischemic heart disease. (154) In contrast, a Japanese study found increased risk of ischemic heart disease for women exposed to ETS. (155)

Scottish researchers found increased mortality rates from heart attacks in men and women exposed to ETS. The researchers did not find elevated levels of coronary heart disease or symptoms in nonsmoking men exposed to passive smoke. However, they did note increased incidence of angina, electrocardiogram abnormality and higher mortality rates for all coronary diseases in nonsmoking women exposed to ETS. (156) These findings were not examined for

statistical significance. Two criticized studies found increased risk for disease from ischemic heart disease (157) and aggravation of angina (158). Considered together, these studies demonstrate risk of heart disease associated with exposure to ETS.

Carcinogenic Effects

Noted authorities, including the U.S. Surgeon General and the National Research Council, have asserted that exposure to environmental smoke increases the incidence of lung cancer for nonsmokers. (131)(134) ETS has been implicated as the cause of 5,000 U.S. lung cancer deaths a year. (159) Most of the studies finding increased risk are epidemiological surveys of nonsmokers exposed to their spouses' smoke. Interpretation of some studies is complicated by methodological problems.

Some investigators have examined the inconsistencies of self-reporting, spouse-reporting, and child-reporting of household smoking habits. One study found that 19% of its self-proclaimed nonsmokers were actually smokers. (160) Another study comparing spouse-reporting and child-reporting of smoking habits demonstrated considerable variability between the two studies. (161) Misclassification of smokers and nonsmokers may thus introduce error in these studies.

Several studies noting significantly increased incidence of lung cancer among spouses exposed to their husband's smoke have been criticized for assuming that nonsmokers' most significant exposure to ETS occurs in the home. This assumption may be incorrect for nonsmoking women in the United States. Nonsmoking Japanese women (162) or Greek women (163), however, are more likely than their American counterparts to work in their homes. Consequently, the women in those two studies may have received their primary exposure to ETS from their husbands, as the studies presumed.

Notwithstanding some of the problems with epidemiological studies to date noted above, strong evidence suggests that ETS is carcinogenic. Several specific points are worth considering at this stage in research on ETS. As mentioned earlier, ETS contains a considerable number of carcinogenic compounds. In addition, mutagenic activity has been noted in several recent studies where filtered ETS particles were applied to bacteria and mutagenic activity was measured. (164)(165) Finally, while inappropriate assumptions and methodological problems may complicate analysis of some epidemiological studies, the weight of evidence indicates an increased lung cancer risk as the Surgeon General and Natural Research Council have acknowledged.

Synergistic Effects

In addition to the increased risk of lung cancer associated with ETS exposure, ETS appears to enhance the effects of other hazardous compounds. For example, "radon-daughters", radioactive radium decay products, are known

to attach to particulates in tobacco smoke. One researcher found radon-daughter concentrations doubled in the presence of ETS. (166) If these submicron or micron sized particles are inhaled, they may expose the host to significant levels of radioactivity.

Asbestos and cigarette smoke are also known to interact. Similar to radon-daughters, submicron sized asbestos fibers may attach to ETS particulates. This increases asbestos fibers' airborne lifespan, which enhances their likelihood of being inhaled. Asbestos is recognized as a cause of lung cancer, mesothelioma, and asbestosis.

Increased Health Risk Posed by ETS Exposure

Public health policy decisions often must be made without certainty as to the magnitude of health benefits resulting from various policy options. Quantitative risk assessment assists public health policy-makers in setting a reasonable upper bound on potential public health risks in the face of such uncertainties. Given the known substantial health risks associated with smoking, health risks associated with ETS exposure require careful examination. Health policy decisions should be based on worst-case risk estimates to best protect public health.

A major ETS risk assessment relative to lung cancer was conducted in 1985 by Repace and Lowrey. (159) This study estimated that aggregate exposure to ambient tobacco smoke produces about 5000 lung cancer deathes per year in U.S. nonsmokers aged 35 years or older, with an average loss of life expectancy of 17 $\pm$ 9 years per fatality. The estimated risk to the nonsmoker most exposed to ETS is comparable to that from pipe and cigar smoking.

MEASUREMENT AND MONITORING OF ETS EXPOSURE

Several methods have been employed to assess ETS exposure, including questionnaires and diaries, exposure modeling, microenvironmental measurements of ETS contaminants, personal measurement of ETS contaminants and biological markers. (167) Each method has distinct benefits and drawbacks which must be considered in evaluating exposure data resulting from their use. The goal of each technique is to assess ETS exposure, with exposure defined as the dynamic integration of concentrations of ETS constituents in various environments and the time an individual spends in each of those environments. (167)

Questionnaires

Questionnaires are often used to obtain the case histories of individuals chronically exposed to ETS. Sometimes, surrogate respondents are used when the subject of the study is deceased. The questionnaires ask questions such

as the smoking status of the respondent, employment history, non-occupational exposure to ETS, length and intensity of exposure to ETS, family health history and, if a smoker, number and type of cigarettes smoked.

Questionnaires do not always account accurately for complex ETS exposure patterns and often are plagued by respondent bias and lack of precision in recall. Still, questionnaires provide the only effective means for obtaining case history information needed to assess the chronic effects of ETS such as lung cancer.

Exposure Modeling

Exposure modeling uses ETS emission rates, dispersion models and time-activity patterns of individuals exposed to ETS. Ventilation in the subject indoor air space as well as the aging and mixing of ETS are factored into a general equilibrium model for ETS. (166) Modeling efforts to date have focused on modeling respirable particulates (RSP) emitted in indoor environments. From the model, RSP levels can be predicted, with these predictions being used to estimate ETS exposure.

Personal and Microenvironmental Exposure

ETS exposure concentrations may be monitored by either personal monitoring or microenvironmental measurements of ETS constituents along with time-activity patterns of the exposed individuals under study. Time-activity patterns refer to the record of the amount of time an individual spends in a variety of places. Measurement of ETS constituent concentrations has occurred on both the microenvironmental and personal levels. A microenvironment is generally an enclosed indoor space where individuals may be exposed to ETS. Personal monitoring of ETS concentration, on the other hand, is derived from the concentration of ETS constituents in an individual's breathing zone. This method is especially useful since it integrates the temporal and spatial dimensions of an individual's exposure to ETS.

Since ETS is a very complex mixture of particulate and gaseous constituents, concentrations of specific "tracer" or "marker" ETS constituents must be measured to assess total exposure to ETS. These tracer components are measured by comparing their concentration in smoking versus nonsmoking environments. Such "tracers" must be unique or nearly unique to tobacco smoke, be present in sufficient quantities to be detected at low smoking rates, have similar emission rates for a variety of tobacco products, and have a fairly consistent ratio to the constituents of interest, such as carcinogenic constituents, over a wide range of environmental conditions and for a variety of tobacco products. (166)

Several ETS constituents have been used as tracers, including carbon monoxide, RSP, nicotine, aromatic hydrocarbons, tobacco-specific nitrosamines, nitrogen oxides, acrolein and acetone. Most of the indoor air monitoring data

base available uses RSP as a tracer, showing a consistently good correlation between smoker occupancy and RSP. However, one drawback for using RSP as a tracer is its nonspecificity for ETS. Therefore, all of the measured RSP does not necessarily originate from ETS, but may be emitted by other indoor sources or enter the building from outside.

Nicotine has also been used extensively as a tracer for ETS. Nicotine has many characteristics of a good tracer, especially since it is specific to ETS exposure; that is, no other nicotine source is known. Problems arise given nicotine's distribution between the particulate and vapor phases. In addition, the ratio of nicotine to other ETS constituents for many tobacco products is unknown.

Carbon monoxide concentrations may indicate exposure to gas phase components of ETS. Carbon monoxide, however, is a poor tracer given its nonspecificity for ETS. Indoor carbon monoxide levels are affected by numerous other sources including stoves, grills and cars.

EPA's recent TEAM study used personal monitoring to develop individual exposure profiles to volatile organic compounds. A significant increase in benzene concentrations was found with individuals exposed to ETS, thus volatile organics may prove to be suitable tracers for ETS exposure. As with carbon monoxide and RSP, however, benzene is not specific to ETS.

Experience with each of these tracers has shown that no single compound can be used to evaluate an individual's exposure to ETS. Still, use of tracers provides valuable information for estimating ETS exposure not gleaned from questionnaires.

Biological Markers

Analysis of human physiological fluids to detect ETS contaminants has been used to assess overall exposure to ETS in individuals. Saliva, urine and blood samples have been used, measuring accumulating levels of thiocyanate, carboxyhemoglobin, nicotine and its metabolite cotinine, hydroxyproline, N-nitrosoproline, aromatic amines and urinary mutagens. Such measurements can provide an estimate of dosage after accounting for the differences in respiratory rates among exposed individuals.

Nicotine and cotinine are currently the best available markers since both of these compounds in biological fluids are highly specific for ETS exposure. Nicotine is highly soluble and upon inhalation is absorbed into the mouth and bronchial tree, ultimately reaching the lung alveoli and bloodstream. Nicotine in ETS appears to be primarily in the vapor phase and may not be directly related to the carcinogenic potential of ETS, thus an indicator for carcinogenic risk is still needed.

Cotinine has a longer half life in blood than nicotine, it is less volatile, and it occurs primarily in the particulate phase. For these reasons, cotinine has become the method of choice for assessing ETS exposure.

The radioimmunoassay method has excellent sensitivity for nicotine and cotinine in bodily fluids and is capable of measuring low level exposure.

Carbon monoxide and carboxyhemoglobin provide another biological marker for ETS exposure. Carbon monoxide is a nonreactive gas produced in the tobacco combustion process. It enters the bloodstream by first penetrating the alveolar regions of the lung. In the blood, carbon monoxide and hemoglobin react to form carboxyhemoglobin, the measurement of which indicates ETS exposure. This marker lacks the sensitivity needed to differentiate between various levels of ETS exposure in addition to carbon monoxide being nonspecific for ETS.

Thiocyanate is a product of the liver's detoxification process for hydrogen cyanide gas and can be measured in blood plasma, saliva and urine. Thiocyanate is also produced in the tobacco combustion process and thus has been used as a potential marker for ETS exposure. Thiocyanate however is nonspecific for ETS since it can be produced from compounds in certain foods. As a result, thiocyanate has been shown not to be an effective measure of ETS exposure. (160)

MITIGATION OF ETS EXPOSURE

A total ban on smoking represents the most efficient means of mitigating ETS exposure. This option is not always perceived as being feasible, therefore, other options have been implemented in these cases. Such options include source modification, air cleaning, dilution ventilation, and exhaust ventilation. (168)

Source Modification

Source modification involves the relocation of smokers to specific smoking areas. As the Surgeon General found in his 1986 report, such a simple separation of smokers and nonsmokers may reduce nonsmoker exposure to ETS but does not eliminate it. (131) This situation is especially true where the smoking areas and nonsmoking areas share a common HVAC system, thus recirculating the smoke throughout both areas. (168)

The use of the new smokeless cigarette might also represent another form of source modification, albeit a poor one. The smokeless cigarette produces little smoke, no tar, no ashes and no smell. In 1987 the Wall Street Journal cited the potential problems in marketing this product, partially due to the fact that no one in the tobacco industry has claimed that the smoke-free cigarette is "safer" or "healthier" than traditional cigarettes. R.J. Reynolds has simply stated that the smoke-free cigarette was designed in part to eliminate some of the ingredients that cause smokers concern. (169)

Air Cleaning

Air cleaning involves an HVAC system that removes ETS from return air before recirculating it throughout the building. Isolated air-cleaning devices may be used near the site of smoking activity. Electrostatic precipitators are effective in removing respirable particulate matter, but not the volatile components of ETS. The use of granulated filter media capable of absorption such as activated carbon or some type of catalytic system has been suggested for the effective removal of ETS volatile components from the airstream. (168)

Dilution Ventilation

Dilution ventilation uses the dilution of ETS contaminated air with uncontaminated air in a general area, room or building to control potential health hazards or nuisances. This type of ventilation does not remedy the fact that surfaces in enclosed rooms act as sinks for ETS particles, and thus ETS odors linger. (168) To minimize such impacts, ASHRAE recommends a ventilation rate of 60 cfm of outdoor air per person in smoking lounges.

Local Exhaust Ventilation

Local exhaust ventilation has been described as providing the greatest potential for obtaining an environment free of ETS. With this method, the collection of ETS-contaminated air occurs as close to the source as possible, minimizing the air volumes required to ventilate the area and maximizing the contaminent collection efficiency. For this type of mitigation, the smoking area must be enclosed and isolated from the HVAC system serving the rest of the building. (168)

Even with all of these possibilities for mitigating ETS in indoor environments, the best remedy is still source elimination. Many efforts to encourage and assist smokers to quit smoking have been made since the 1960s. For example, federally-sponsored television advertisements described the health effects of cigarette smoking. Corporations have developed in-house smoking cessation programs. More recently, innovative approaches designed to help the smoker quit have been developed. Nicotine gum is one such innovation. A newer technique makes the smoker inhale a mist made from an irritating chemical found in citrus fruits (citric acid), which substitutes for the "taste" of tobacco. (170) This therapy may be coupled with another therapy whereby nicotine is absorbed through the skin from a stick-on patch. (170)

INTERNATIONAL STATUS OF ETS POLICY

Smoking and ETS exposure are recognized as significant health threats worldwide. Numerous studies have been conducted regarding passive smoking in Japan and the United Kingdom. Regulatory restrictions and isolated bans have been instituted.

In the United Kingdom, a March 1988 study suggested that ETS causes several hundred lung cancer deaths per year in the U.K. The study also states that "improved ventilation, or the mixing of smoking and nonsmoking areas within the same enclosed space would not seem to provide adequate safeguards" for nonsmokers. (171) The report concludes by suggesting that a ten to thirty percent increase in lung cancer risk exists for nonsmokers exposed to ETS. (171)

Such studies prompted the British Petroleum Industry to restrict smoking activity in its headquarters. Beginning in November 1988, smoking is completely banned on the London-Glasgow shuttle on a trial basis. (172)

Canada's Federal-Provincial Advisory Committee on Environmental and Occupational Health released a report in April 1987, establishing exposure guidelines for residential indoor air quality. (173) The primary objective of the report was to develop these guidelines to serve as a reference point against which the adequacy of residential indoor air quality and remedial measures can be judged. The Advisory Committee's singular recommendation for tobacco smoke:

> "In view of the carcinogenic properties of tobacco smoke, it is recommended that any exposure to tobacco smoke in indoor air environments be avoided." (173)

The Sixth World Conference on Smoking and Health took place in Tokyo in November 1987. Some of the more significant conference recommendations include:

(1) All countries should work toward establishing smoke-free environments in all enclosed public places; worksites; and transportation, health care, child care and school facilities.

(2) Tobacco control legislation should be viewed as an integral part of a comprehensive tobacco control policy.

(3) The Conference endorsed the legitimacy of claims by victims of tobacco-induced disease to obtain compensation from tobacco manufacturers.

(4) Socio-economic and health costs to both the individual and society as a whole should be assessed. Appropriate tax rates and insurance rates should then be adjusted, reflecting the true cost of smoking to society.

(5) The issue of women and tobacco should be integrated into local, national and international tobacco control strategies.

(6) Increased international cooperation is needed in research of tobacco use by children.

(7) Every country should recognize the complex nature of tobacco use and should implement appropriate and demonstrably effective cessation techniques.

(8) Tobacco advertising, sponsorship and any other forms of direct or indirect promotion should be banned in all countries. (170)

FEDERAL ETS STUDIES

The Surgeon General's 1986 report entitled "The Health Consequences of Involuntary Smoking" catalyzed the mounting concern for nonsmokers exposed to ETS. In his report, the Surgeon General explores the health effects of ETS and the policies restricting smoking in public places and the workplace. As a result of his review, the Surgeon General made the following conclusions:

> (1) Involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers.
>
> (2) The children of parents who smoke compared with the children of nonsmoking parents have an increased frequency of respiratory infections, increased respiratory symptoms, and slightly smaller rates of increase in lung function as the lung matures.
>
> (3) The simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate the exposure of nonsmokers to environmental smoke. (131)

Just one month before the release of the Surgeon General's report, the National Academy of Sciences released its study of children of smokers entitled "Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects." The Academy concluded that children and spouses of smokers face a higher risk of respiratory ailments than those of nonsmokers. The risk of a smoker's child suffering from wheezing, coughing and the production of sputum is 20-80% greater than for the child of a nonsmoker. The Academy found that spouses of smokers have a 30% higher chance of getting lung cancer than those of nonsmokers. Citing radon's ability to absorb to respirable particulate matter in tobacco smoke, thus allowing more efficient inhalation of the radon, the Academy recommended further study of radon and tobacco constituent interactions.

In June of 1987, the EPA issued its "EPA Indoor Air Quality Implementation Plan" in which its ongoing indoor air research programs are described. Several studies focus on ETS:

(1) Evaluation of Field Methods to Estimate Exposure in Epidemiological Studies.

(2) Personal Activity Related Exposure to ETS in Airliner Cabins and Other Transporation Related Environments.

(3) Evaluation of Sampling and Analytical Methods for Nicotine and PAHs.

(4) Biological Markers for ETS Human Exposure Assessment.

(5) Development of Biological Markers for Molecular Dosimetry Resulting from Exposure to ETS.

(6) Evaluation and Improvement of Cotinine as a Biological Marker of ETS Exposure in Children and Adults.

In May 1988, the nineteenth volume of the Surgeon General's reports on the health consequences of smoking was issued. In this report, Surgeon General C. Everett Koop discusses the addictive nature of nicotine, likening its addictive qualities to those of illegal substances such as heroin and cocaine. (174)

Surgeon General Koop announced specific ideas for new legislation, primarily geared toward discouraging children and teenagers from ever starting to smoke. These ideas include a proposal to require labels on tobacco products notifying the potential buyer of nicotine's addictive qualities. Koop further suggests that cigarette excise taxes should be increased. Koop also recently unveiled an anti-smoking public service message targeting children which says "Don't be a butthead." Koop has also called for a ban on vending machine sales of cigarettes and stricter regulations on the distribution of free cigarette samples. (174)

FEDERAL REGULATION OF ETS

Federal regulation of ETS is sparse in contrast to extensive state and local controls of ETS. Applicable federal regulations on ETS are summarized below.

Department of Transportation, 14 CFR Part 252

"Smoking Aboard Aircraft"

These regulations establish rules for regulating smoking aboard aircraft, including the designation of smoking areas and prohibition of smoking when the aircraft is on the ground, when the ventilation system is not fully functional, and when passenger capacity is less than thirty (30) seats. Cigars and pipes are prohibited on all aircraft.

Interstate Commerce Commission, 49 CFR Part 1061

"Limitation of Smoking on Interstate Passenger Carrier Vehicles"

These regulations provide for separate seating for smokers and nonsmokers on interstate passenger carriers by motor vehicles. Smoking is permitted only in the rear of the vehicle and may not exceed 30% of vehicle capacity.

Department of Defense, 32 CFR Part 203

"Smoking in DoD Occupied Buildings and Facilities"

These regulations control smoking in DoD-occupied buildings, and facilities. Smoking is prohibited in auditoriums, conference rooms, classrooms and shuttle vehicles. Smoking is restricted in medical care facilities and allowed in private offices and in common work areas only if ventilation is adequate to remove smoke from a work area and provide an environment that is healthful. Smoking is permitted in restrooms, lobbies and corridors.

General Services Administration, 41 CFR Part 101-20, 105-3

"Smoking"

These regulations control smoking in GSA-controlled buildings and facilities, including leased areas and delegated spaces. The regulations recognize the increased health hazard of passive smoke on the nonsmoker. Smoking is prohibited in auditoriums, classrooms, conference rooms, elevators, medical care facilities, and hazardous areas. Smoking is also prohibited in corridors, lobbies, restrooms except if a sufficient number of other smoking areas are unavailable. Cafeterias must have smoking areas and office space may be designated a smoking area as long as it is configured and ventilated so as to protect the nonsmokers against involuntary exposure to smoke. No expenditures are required by the agency to accommodate the preference of nonsmokers.

A federal ban on smoking on all airplane flights of two (2) hours or less began on April 23, 1988 for an experimental two (2) year period. This ban effects 80% of all U.S. flights. (170)

The 1989 U.S. Congress will be considering at least three bills introduced in the last session pertaining to ETS, including the following:

(1) HR1008, originally submitted February 4, 1987.
To protect the health of nonsmokers working and visiting in government buildings by restricting smoking to designated areas in all buildings occupied by federal employees.

(2) HR5394, originally submitted September 27, 1988.
To provide for a total ban of smoking in all hospitals participating in the Medicare/Medicaid programs as a criterion for maintaining eligibility for these programs.

(3) S51, originally submitted January 6, 1987.
To prohibit smoking in all public conveyances. (175)

Former Secretary Caspar Weinberger of the Department of Defense issued an initiative restricting smoking to designated areas on all military property and establishing an educational program. Current Secretary Carlucci has proposed to strengthen the military's anti-smoking policy by raising the price of cigarettes in military stores close to the price paid by civilian consumers. The sale of cigarettes would also be prohibited in military commissaries (supermarkets) and permitted only in military exchanges (drugstores).

CONTROL OF ETS IN MASSACHUSETTS

Massachusetts Laws on ETS

Massachusetts recently passed legislation (M.G.L. c. 270, Sec.22) restricting smoking in many public places. The law augments and expands upon existing laws that restrict smoking in public conveyances, nursing homes, town meetings, school buses, primary and secondary schools, jury rooms and selected dormitory rooms at public universities.

Table 6-2 lists those areas in Massachusetts where smoking is prohibited and where smoking is restricted to designated areas. The owners, managers or other persons in charge of any of the buildings or facilities in Table 6-2 where smoking is only allowed in designated areas must post conspicuous notices as to which are the smoking and nonsmoking areas. A smoking area can be designated only if there is an area large enough for nonsmokers. The law does not require that smoking be allowed.

The Massachusetts Plan for Nonsmoking and Health

The Massachusetts Department of Public Health (DPH) completed its Plan for Nonsmoking and Health in Massachusetts in September 1988. (132) The plan establishes goals for the reduction in tobacco use and increase of ETS control. Specifically, by the year 1995, the plan calls for more than 90% of all females of childbearing age to be nonusers of tobacco, more than 85% of low income adults to be nonusers of tobacco, and less than 10% of adults to be heavy smokers.

Table 6-2

Does state law limit smoking?

State law prohibits smoking in:

- public elevators
- retail food outlets
- polling places
- school buses
- open meetings of governmental bodies
- supermarkets
- courtrooms
- town meetings
- public mass transit vehicles and enclosed indoor or outdoor waiting areas

Smoking is allowed only in designated areas in:

- museums
- hospital lobbies
- trains
- restaurants with 75 or more seats
- colleges and universities
- group child care centers
- public buildings
- libraries
- nursing homes
- airplanes
- courthouses
- airport waiting areas
- school-aged day care centers
- schools

(adapted from the Mass. DPH Fact Sheet on ETS)

By the year 1995, the plan also calls for all Massachusetts schools, public places and health care facilities to be smoke free and all businesses to be smoke free except for designated areas. The DPH's goal for the year 2000: more than 90% of adults and adolescents in Massachusetts will be nonusers of tobacco and nonsmokers will be able in the course of their normal daily activities to breathe ETS-free air.

In order to achieve these goals, the DPH recommends widespread efforts to educate especially young people and provide free information about the health effects of smoking and ETS exposure, including cessation programs to assist smokers to quit permanently.

The DPH also recommends areas where legislation will be critical in the achievement of the plan's overall goals. These specific recommendations include:

- Laws prohibiting tobacco sales to minors should be strengthened, including the enforcement provisions of such laws, and regulations regarding sale of tobacco products through vending machines should be promulgated.

- Laws should be enacted, prohibiting tobacco promotion and advertising generated within Massachusetts. In particular, the law should prohibit free distribution of tobacco, event sponsorship and billboard advertising.

- A law should be enacted prohibiting tobacco use by all persons in all public and private schools, or school grounds, and at school-sponsored events.

- Existing laws and regulations restricting smoking in schools, health care facilities, businesses and other public places should gradually be strengthened.

- All Massachusetts businesses should be nonsmoking or have policies restricting smoking to designated areas that are physically separate from work and common areas.

- All new forms of manufactured tobacco or tobacco-like products such as the "smokeless cigarette" should be subject to the same health and consumer safety laws that apply to all other products and prohibited from sale if they fail to meet standards for health and safety.

Local Regulation of ETS

Local Boards of Health in Massachusetts have broad authority to promulgate reasonable health regulations for ETS in schools, restaurants, government buildings and many other places. A total of fifty-one communities in Massachusetts have laws guaranteeing nonsmoking sections in restaurants. (176) Nine communities surpass state requirements by protecting nonsmokers in the private sector workplace.

STATE AND LOCAL REGULATION OF ETS ACROSS THE NATION

Table 6-3 provides an overview of state smoking laws across the country as of 1986. The laws referenced are similar in substance to those in Massachusetts. Since 1986, several states and localities have taken more progressive steps in controlling nonsmokers' exposure to ETS.

Table 6-3

State laws regulating smoking in public places and worksites (1)

State	AL	AK	AZ	AR	CA	CO	CT	DE	DC	FL	GA	HI	ID	IL	IN	IA	KS	KY
Year(s) legislation enacted	—	1975 1984	1973 1981	1977 1985	1971,76 1980,81 1982	1977 1985[1]	1973 1974 1983	1960	1979	1983 1985	1975	1976	1925 1975 1985	—		1978	1975	1972
PUBLIC PLACES WHERE SMOKING IS PROHIBITED (EXCEPT IN DESIGNATED AREAS)																		
Public transportation		X	X	(X)[2]	X[3,4]	X	X[3]	X	X	X[3,5]	X[5]		X			X	X	X
Elevators		X[5]	X			X	X[5]		X	X[5]	X[5]	X	X			X	X	
Indoor recreational or cultural facilities[6]		X	X		X	X				X		X	X			X	X	
Retail stores		(X)[7]			(X)[7]	X	X		X	X			X					
Restaurants		X[8]			X[9]	X	X[10]			X[6]			X					
Schools		X	X	X	X	X	X		X	X			X					X
Health care facilities																		
Hospitals		X	X	X	X	X	X		X	X		X	X			X	X	
Nursing homes		X			X	X	X		X	X			X			X	X	
Public meeting rooms		X			X		X		X	X		X	X			X	X	
Libraries		X	X							X						X	X	
Restrooms		X[5]	X			X	X			X								
Waiting rooms		X	X			X	X			X						X		
Other		X[26,27]	X[27]							X[26,27,30]								
WORKSITE SMOKING RESTRICTIONS[16]																		
Public worksites		D[17]			B	D[1]	B		B	B,D[16]		B[1]	B			D		
Private worksites		A					B			B,D								
IMPLEMENTATION PROVISIONS																		
Nonsmokers prevail in disputes		X																
No discrimination against nonsmokers																		
ENFORCEMENT																		
Penalties for violations		X	X	X	X		X	X	X	X	X	X	X			X	X	X
Smoking		X[23d]	X[23p]	X[23e]	X[23e]		X[23c]	X[23c]	X[23e]	X[23i]	X[23e]	X[23e]	X[23d]			X[23e]	Z[23c]	X[23e]
Failure to post signs		X[24h]							X[24h]									
Overall State law restrictiveness:[25]	0	3	2	1	3		3	4	1	2	4	1	2	3	0	0	2	2	1

State	LA	ME	MD	MA	MI	MN	MS	MO	MT	NE	NV	NH	NJ	NM	NY	NC	ND	OH
Year(s) legislation enacted	—	1954 1981,83 1985	1957 1975	1924 1947 1975	1967 1968 1978	1971 1975	1942	—	1979	1979	1911 1975	1981	1955 1979 1985	1985	1921 1975 1976	—	1977	1981 1984
PUBLIC PLACES WHERE SMOKING IS PROHIBITED (EXCEPT IN DESIGNATED AREAS)																		
Public transportation		X	X	X[5]	(X)[2]	X	X		X	X	X	X	X	X	X		X	X
Elevators			X	X[5]	X	X			X	X	X	X	X	X			X	X
Indoor recreational or cultural facilities[6]				X		X			X	X	X	X	X	X	X		X	X
Retail stores		X		(X)[7]	X	X			X	X		X	X					
Restaurants					X	X[11]			X	X			X[12]	X[12]			X	
Schools						X			X	X	X	X	X	X			X	X
Health care facilities																		
Hospitals		X	X	X	X	X			X	X	X	X	X	X			X	X
Nursing homes		X	X	X	X	X			X	X	X	X	X	X			X	X
Public meeting rooms						X			X	X	X	X	X	X			X	X
Libraries				X		X				X	X		X	X	X		X	
Restrooms		X				X				X							X	X
Waiting rooms		X				X				X	X	X					X	X
Other		X[26]		X[28]		X[26,27]				X[27]		X[29]			X[29]			
WORKSITE SMOKING RESTRICTIONS[16]																		
Public worksites		B,D				D[17]			D[19]	D[17]		D[20]	B,C[17]	B,C[20]			C	C
Private worksites		B,D				D[17,21]			D[19]	D[17,21]	A[22]		B,C[17]		A[22]			
IMPLEMENTATION PROVISIONS																		
Nonsmokers prevail in disputes						X								X				
No discrimination against nonsmokers		X																
ENFORCEMENT																		
Penalties for violations		X	X	X	X	X	X		X	X	X	X	X	X	X		X	X
Smoking		X[23d]	X[23h]	X[23j]	X[23k]	X[23n]	X[23o]			X[23e]	X[23e]	X[23l]	X[23g]	X[23c]	X[23e]		X[23e]	X[23m]
Failure to post signs		X[24e]			X[24e]				X[24c]				X[24g]					
Overall State law restrictiveness:[25]	0	4	2	2	3	4	1	0	4	4	2	2	4	3	2	0	3	2

Table 6-3 (cont'd)

State	OK	OR	PA	I..	SC	SD	TN	TX	UT	VT	VA	WA	WV	WI	WY	Total
Year(s) legislation		1973,75	1927										1913			N (%)
enacted	1975	1977 1981	1947 1977	1976 1977	1937	1974	—	1975	1976 1979	1892	—	1983	1919 1985	1984	—	51 (100)
PUBLIC PLACES WHERE SMOKING IS PROHIBITED (EXCEPT IN DESIGNATED AREAS)																
Public transportation	X			X	(X)[2]	X		X	X			X[5]	X	X		35 (68.6)
Elevators	X	X		X		X		X	X			X[5]		X		31 (60.8)
Indoor recreational or cultural facilities[6]	X	X	X	X		X		X	X			X[5]		X		29 (59.6)
Retail stores		X	X	X					X			X[5]		X		18 (35.3)
Restaurants		X		X					X			X		X		18 (35.3)
Schools		X		X		X		X	X			X[5]	X	X		27 (52.9)
Health care facilities																
Hospitals		X	X	X		X		X	X			X		X		33 (64.7)
Nursing homes		X	X	X				X	X			X		X		29 (56.9)
Public meeting rooms		X							X			X[5]				21 (41.2)
Libraries	X			X		X		X	X			X				19 (37.2)
Restrooms												X				11 (21.6)
Waiting rooms		X										X		X		16 (31.4)
Other			X[30]	X[28]								X[15]				12 (23.5)
WORKSITE SMOKING RESTRICTIONS[16]																
Public worksites		D							D[17]			D		D[18]		22 (43.1)
Private worksites									D[17,22]	A[22]		D	A[22]			9 (17.6)
IMPLEMENTATION PROVISIONS																
Nonsmokers prevail in disputes									X							4 (7.8)
No discrimination against nonsmokers									X							2 (3.9)
ENFORCEMENT																
Penalties for violations	X	X	X	X		X		X	X	X		X	X	X		40 (78.4)
Smoking	X[23e]	X[23b]	X[23c]	X[23e]		X[23p]		X[23o]	X[23o]	X[23a]		X[23f]	X[23a]	X[23c]		39 (76.5)
Failure to post signs		X[24e]		X[24e]									X[24e]			9 (17.6)
Overall State law restrictiveness:[25]	2	3	2	3	1	2	0	2	4	1	0	4	1	3	0	

[1] Executive order.
[2] School buses only.
[3] Including school buses.
[4] California stipulates that at least 50 percent of all passenger seats must be in nonsmoking areas on trains, airplanes, and street railroad cars departing from the State.
[5] Smoking never permitted in this area.
[6] Indoor recreational and cultural facilities: museums, auditoriums, theaters, and sports arenas.
[7] Grocery stores only.
[8] Restaurants seating 50 or more persons must have a no-smoking section.
[9] Restaurants seating 50 or more persons must have a no-smoking section if the restaurant is in a publicly owned building.
[10] Restaurants seating 75 or more persons must have a no-smoking section.
[11] Restaurants must designate at least 30 percent of their seats as a no-smoking area.
[12] Restaurants are encouraged to establish no-smoking areas.
[13] Restaurants must designate at least 50 percent of their seats as a no-smoking area.
[14] (Deleted).
[15] No place other than a bar may be designated a smoking area in its entirety.
[16] Worksite (only B, C, and D count as having a worksite policy in caculation of totals): A - Employer must post a sign prohibiting smoking at the worksite; B - Employer must have a (written) smoking policy; C - Employer must have policy that provides a nonsmoking area; D - No smoking except in designated areas.
[17] Employer must post signs designating smoking and no-smoking areas.
[18] Employer must post signs in smoking areas.
[19] Employer must post either smoking or no-smoking signs, depending upon their policy.
[20] Employer must post signs in no-smoking areas.
[21] State does not restrict smoking in factories, warehouses, and similar places of work not usually frequented by the general public.
[22] Prohibits smoking in any mill or factory in which a no-smoking sign is posted.
[23] Persons who smoke in a prohibited area are subject to a fine or a penalty. Maximum fines or penalties, where applicable, are listed below: a = $5; b = $10; c = $25; d = $50; e = $100; f = $100/day; g = $200; h = $300; i = $500; j = $50 or up to 10 days in jail or both; k = $50 or 90 days imprisonment; l = civil action; m = minor misdemeanor; n = petty misdemeanor; o = misdemeanor; p = petty offense.
[24] Persons who are required to and fail to post smoking and/or no-smoking signs are subjected to a penalty. Maximum fines, where applicable, are listed in footnote 23.
[25] Restrictiveness key: 0 = None, no statewide restrictions; 1 = Nominal, State regulates smoking in one to three public places, excluding restaurants and private worksites; 2 = Basic, State regulates smoking in four or more public places, excluding restaurants and private worksites; 3 = Moderate, State regulates smoking in restaurants but not private worksites; 4 = Extensive, State regulates smoking in private worksites.
[26] Jury rooms.
[27] Halls and stairs.
[28] Stables.
[29] Polling places.
[30] Prisons, at prison officials' discretion.

New York passed a "Clean Indoor Air Act" on April 1, 1988. This act requires employers with more than fifteen employees to write a smoking policy within ninety days of the Act's effective date. Employees are permitted to designate their workplace as a nonsmoking area if they choose. (176) Nassau County, New York prohibits smoking in places of employment entirely, except in totally enclosed offices occupied by smokers and sections of cafeterias, lounges and lunchrooms that are physically separate from work areas and take up no more than 25% of capacity. (176)

California's Proposition 65 Scientific Advisory Panel reported that ETS should be recognized as "known to the state of California to cause cancer." (177) The panel also called ETS a reproductive toxin which affects the reproductive abilities of men and women and is associated with low birth weight. Proposition 65 was adopted in November 1986 and requires clear and reasonable danger warnings to consumers, customers, and employees exposed to toxic chemicals. As a result of Proposition 65, it has been suggested that building owners who expose people to ETS without warnings have the burden of proof to show that ETS is not a significant cancer risk.

By early 1988, California had a total smoking ban in place on all forms of public transportation, including buses, trains, ferries and airlines. A survey taken at San Francisco International Airport revealed that 85% of the airline passengers (22% of whom were smokers) approved of the smoking ban. Of the airline crew members, 94% (13% of whom were smokers) approved of the ban and explained that they felt better at the end of the day when no smoking was permitted. (172)

In March 1988, Montgomery County, Maryland put in place extensive smoking bans. Smoking is prohibited in public restrooms, enclosed auditoriums, and public areas of retail stores, banks, offices, factories and any other private business. Montgomery County already had laws prohibiting smoking in elevators, schools, health care facilities, county government offices, theatres, county workplaces, and rail transit stations. (178)

Rhode Island recently passed the "Workplace Smoking Pollution Control Act." This act requires employers to develop smoking policies and to attempt to reach a reasonable accommodation in so far as possible, between the preferences of smokers and nonsmokers. All workplaces are regulated by this act except private homes, offices leased or rented by one person or private enclosed workplaces occupied only by smokers. Employers who violate this act are subject to fines.

San Francisco was the first city to ban workplace smoking in 1983. Likewise, the University of California in San Francisco became the first smoke free UC campus on June 6, 1988. In Sanoma County, California, a local ordinance adopted in June 1988 gives priority to nonsmokers by permitting them to designate nonsmoking areas.

CORPORATE APPROACH TO CONTROLLING ETS EXPOSURE

While federal, state and local public officials grapple with the problem of ETS exposure, U.S. corporations are developing their own smoking policies, with a trend toward stricter policies. A 50% increase in the number of companies restricting smoking has been reported, from 36% in 1986 to 50% in 1987. (170) The three main reasons given by corporations for adopting such policies are, in order of importance, employee complaints, concern about employee's health, and state and local laws. (179) Other reasons cited include fear of lawsuits, worry about the need to compensate nonsmokers, public image, recognition that there is no legal right to smoke on the job, and concerns of customers. (170) Smoking policies are especially important in industries where workers are exposed to asbestos and other toxic substances which have been shown to have a synergistic effect with smoking.

Corporate policies may be grouped into the following general categories:

Policy A

It is the policy of the company to hire nonsmokers only. Smoking is prohibited off the job, and smoking is prohibited on the job.

Policy B

Smoking is prohibited in all areas on company premises.

Policy C

Smoking is prohibited in all areas in company buildings.

Policy D

Smoking is prohibited in all areas in company buildings, with few exceptions. Smoking is permitted in the smoking section of the cafeteria (or room with a similar function in a company if there is no cafeteria); in specially designated smoking rooms (smoking lounges); and in private offices, which may be designated "smoking permitted" or "no smoking" by the occupant.

Policy E

Smoking is prohibited in all common areas except those designated "smoking permitted."

Smoking is permitted in specially designated smoking rooms (smoking lounges).

In open offices and in shared workspace areas where smokers and nonsmokers work together and their preferences are in conflict, employees and management will endeavor to find a satisfactory compromise. On failure to find a compromise, the preference of the nonsmoker will prevail.

Private offices may be designated "smoking permitted" or "no smoking" by the occupant.

Policy F

It is the policy of the company to respect the preferences of both smokers and nonsmokers in company buildings. Where smokers' and nonsmokers' preferences are in conflict, employees and management will endeavor to find a satisfactory compromise. Upon failure to reach a compromise, the preferences of the nonsmoker will prevail.

Policy G

The company places no restrictions on employee smoking (the company does not have a smoking policy). (179)

These policy categories were developed by J. Carroll Swart as part of a study focused on identifying existing (1987) and predicted smoking policies in U.S. corporations through the use of questionnaires. Questionnaires were distributed to banks, data processing firms, savings and loan associations, utilities and insurance carriers.

Sixty-eight percent (68%) of the questionnaire respondents had some type of smoking policy; thirty-two percent (32%) had none. Of the 68%, the distribution among smoking policy types was identified and is shown in Figure 6-4.

These same companies also predicted what their company policy would be in the years 1990 and 1995. A shift is predicted toward the more restrictive policies A, B and C. So far in 1988, Ralston Purina Company and Allstate Insurance Company, both Fortune 500 companies, and Northwest Airlines have instituted total smoking bans. (179) (180) (181) However, Policy D is expected to remain the most prevalent as it was shown to be in 1987. Even companies currently without any type of smoking policy predicted some future restrictions, with Policy F predominating in 1990 and Policy E in 1995.

ECONOMIC COSTS ATTRIBUTABLE TO SMOKING

Economic reasons for restricting smoking have been advanced in recent years. The Massachusetts Department of Public Health (DPH) conducted a study that estimated smoking-attributable costs in Massachusetts for the year 1985. (182) Table 6-4 presents a summary of DPH's cost estimates.

The DPH calculated 87,073 lost years of life due to smoking-induced premature death. These lost years translate into 462 million dollars in lost potential earnings in 1985.

In states with high unemployment rates, this void could be filled easily, thus limiting the economic impact of smoking-related deaths. The unemployement rate in Massachusetts is quite low, therefore this loss in earning potential is of significant concern. Potential earnings are also lost due to debilitating but non-fatal or slowly fatal smoking-induced disease. The DPH estimates 288 million dollars were lost in 1985 for this reason.

The DPH estimated direct health care costs for smoking-induced disease to be 847.6 million dollars. Adding this amount to the loss in potential earnings, smoking-attributable costs for Massachusetts totaled 1,597 billion dollars in 1985.

Smoking-related costs substantially impact private businesses. It has been suggested that employers save $4500 per year for every employee that quits smoking. This savings results from less absentee days and a reduction in morbidity and premature mortality, insurance costs, lost time on smoking rituals and property damage and depreciation. (183)

Productivity may also be affected by cigarette smoke's irritant effects and its ability to depress the central nervous system. A 1985 Office of Technology Assessment study estimated costs included in lost productivity factors and higher health costs alone to be $300-$800 annually. (184)

OPPORTUNITY FOR RESTRICTING ETS EXPOSURE IN PUBLIC AREAS AND WORKPLACES

As awareness of the ETS-induced health effects has grown, private employers and legislators have experienced increased pressure to provide tobacco-free work environments and public areas.

As noted earlier, a trend toward smoke-free work environments is occurring. The Washington Supreme Court recently heard a case brought by an employee seeking damages from her employer for its breach of duty to provide a smoke-free workplace. All nine justices agreed with a state appellate court holding that the employee had stated a claim that would entitle her to relief if she could prove it and therefore remanded it for trial. A minority of the

TABLE 6-4

Summary of Smoking-Attributable Costs

Massachusetts, 1985

Age/Sex	Direct Costs [1]	Indirect Mortality [2]	Indirect Morbidity [3]	Total Costs
Ages 20+				
Both Sexes	$847,548,429	$462,001,173	$288,350,142	$1,597,899,744
Male	$466,357,874	$352,028,076	$192,707,597	$1,011,093,547
Female	$381,190,566	$109,973,097	$ 96,782,638	$ 587,946,286
Ages 20 to 64				
Both Sexes	$513,832,826	$395,060,415	$274,496,405	$1,183,389,647
Male	$321,178,955	$311,719,662	$183,070,145	$ 815,968,763
Female	$192,653,871	$ 83,340,753	$ 93,706,153	$ 369,700,777
Ages 65+				
Both Sexes	$333,715,603	$ 66,940,758	$ 13,853,737	$ 414,510,098
Male	$145,178,919	$ 40,308,413	$ 9,637,452	$ 195,124,784
Female	$188,536,684	$ 26,632,345	$ 3,076,480	$ 218,245,509

1 Direct Costs are expenditures for the prevention, diagnosis, and treatment of smoking-related diseases and medical conditions.

2 Indirect Mortality Costs are the estimated costs of lost income and productivity resulting from premature death due to smoking-related disease and injury.

3 Indirect Morbidity Costs are the costs of lost income and productivity for persons who are disabled by nonfatal or slowly fatal smoking-related illness.

(Shultz, James M., Ph.D. et al., Office of Nonsmoking and Health, Massachusetts Department of Public Works, 1985)

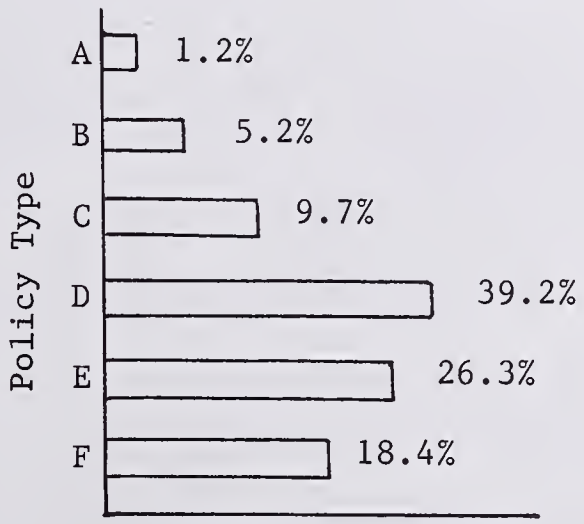
justices believed that the employer's common law duty to provide a safe workplace includes a duty to provide a working environment reasonably free from tobacco smoke. (185)

A 1976 case, Shimp v. New Jersey Bell Telephone Co. involved an employee seeking a smoke-free work environment due to a physical aversion to ETS. The court ruled that an employer must restrict employee smoking to non-work areas if an employee is adversely affected by cigarette smoke. The court also cited the Occupational Safety and Health Act and its requirement for employers to provide a reasonably safe environment and to eliminate all foreseeable and preventable health hazards. A similar court holding resulted in Smith v. Western Electric Company. In both cases, the employees had to prove not only the health threat ETS posed for them individually, but also to the employees in general.

The judiciary has demonstrated a willingness to uphold ETS restrictions both in the workplace and in public areas as long as a rational basis for such restrictions is provided. (184)(185) Although the courts have not yet addressed total smoking bans in workplaces, employers creating such a restriction would represent a legitimate action given the employer's basic responsibility to provide a safe work environment.

FIGURE 6-4

Type of Policy Among
Companies with a Smoking
Policy in 1987



(Source: J. Carroll. Personnel, August 1988, p. 61.)

Chapter 7: BIOLOGICALS

TYPES OF BIOLOGICAL CONTAMINATION

Many biological contaminants are present in indoor environments, including molds, mold spores and toxins, bacteria, viruses, protozoans, algae, body parts and excreta of insects, acarids and arachnids, dander and excreta from animals, pollens from higher plants and house dust. (87) The National Institute for Occupational Safety and Health ranks microbes third among important indoor air problems, after poor ventilation and building fabric contaminants. (187) Biological contaminants may cause infections, allergies and in extreme cases toxic poisoning and death.

Bacterial and viral contaminants may be transported indoors by people or by exhalation from the respiratory tract by breathing, coughing, or sneezing. (84) Plants, pets and insects are potential sources of pollen, dander and other types of allergens. Bacteria and fungi, including molds, mold spores, mildews and actinomycetes may grow and flourish in improperly maintained air ducts, air conditioners, humidifiers, dehumidifiers, air cleaning filters and carpets. (188) Poor ventilation and high humidity levels have been implicated as potential causes of high indoor concentrations of these biological contaminants. Table 7-1 lists various biological contaminants which may be found in indoor environments.

HEALTH EFFECTS OF BIOLOGICAL CONTAMINANTS

Biological contaminants include the following types relative to health effects:

(1) allergenic, i.e. capable of inducing an allergic response which may be characterized by such symptoms as sneezing, itching, skin rashes, asthma, malaise and headaches.

(2) infectious, i.e. causing or capable of causing disease.

(3) toxicogenic, i.e. producing toxic or poisonous products.

Allergic Responses to Indoor Contaminants

Individuals suffering from allergies are predisposed to secrete large amounts of an antibody when exposed to an allergen to which they are sensitive. House dust, dust mites, plant pollens, roach fecal pellets, insect and arachnid dried hulks and body parts, animal dander, amoebae, algae, fungi

and mold spores act as allergens. An antigen-antibody reaction, or allergic reaction, may occur anywhere on the skin, or in the nose, airways, or alveoli of the lungs. Specific effects on body tissues include the dilation of blood vessels, mucous secretion, contraction of smooth muscles lining the bronchial

TABLE 7-1

INDOOR BIOLOGICAL CONTAMINANTS

Fungi and Fungal Spores

Cladosporium	Aureobasidium
Penicillium	Fusarium
Alternaria	Yeasts
Aspergillus	Sporobolomyces
Geotrichum	Wallemei

Bacteria

Legionella pneumophila	Salmonella typhimurium
Clostridium perfringens	Mycobacterium
Staphyllococcus aureus	Pseudomonas
S. epidermidis	Actinobacter
Streptococcus pyogenes	Thermophilic Actinomycetes

Viruses

Smallpox	Influenza
Chicken pox	Adenovirus 4 and 7
Measles	Coxsackie AZ1
Rubella	Lymphocytic Choriomeningitis

Protozoans

Algae

House dust and dust mites

Dander and Excreta from Animals and Insects

Pollens

airways of the lungs, and cellular inflammation. Resultant symptoms include stuffy or runny noses, sneezing, coughing, itching, skin rashes, malaise, difficulty in breathing and headaches. (84)

Asthma is believed often to be caused by allergens. Asthma may be defined generally as a condition of intermittent wheezing caused by variable and irreversibly widespread narrowing of bronchial airways. (189) Asthma caused by specific allergens is referred to as extrinsic asthma. The term "intrinsic" designates cases of asthma with no identifiable cause but often associated with infections, weather changes, exposure to cold, exercise and emotional upset. About 3 to 5% of the population suffer from asthma. (84)

Table 7-2 shows the paramount role house dust and pollen play in causing extrinsic asthma. Clinical studies have provided convincing evidence of house dust mite inhalation causing asthma. (190) Indoor pollen concentrations are driven by outdoor concentrations, with the pollen entering through building cracks, doors, windows and the fresh air intake of air conditioning systems. (87) Animal dander also represents a frequent cause of allergy. (189)

TABLE 7-2

Dominant Allergens in the Cause of Extrinsic Asthma (1970-1980)

Age Group (yrs)	Total	House Dust Group	Pollens and fungi spores	Miscellaneous
0-4	1010	750	17	243
5-14	851	589	183	79
15-29	2096	784	1241	71
30-44	801	356	403	42
45-59	162	86	44	32
60 plus	13	8	3	2
total	4933	2575 (52.2%)	1889 (38.3%)	469 (9.5)

Note: The Miscellaneous group includes Epidermals, foods, drug insect reactions ets.
Source: Ford, R.M., M.D. Annals of Allergy, Vol. 50, Jan. 1983.

One of the most common types of allergies caused by these substances is allergic rhinitis, or hay fever, which afflicts about 15% of the population. (189)(84) In hay fever, asthmatic symptoms include nasal air passage obstruction and itching, sneezing, oversecretion of mucous, and often conjunctivitis. Asthma represents another type of allergy which occurs in 7% of the population and is characterized by episodes of cough, shortness of breath and wheezing.

Molds and mold spores also play an important role in allergic diseases, especially in children. (191) Molds originate primarily in soil and water and

are often carried into the home by persons entering with mold spores on their clothes and shoes. Secondary sources of indoor molds are paper products, wallpaper paste, paint, wood, natural and synthetic fibers and leather. Furniture and mattress dust and stuffed animal toys harbor molds. Moist surface and water reservoirs for air conditioning and humidifying systems can serve as reservoirs and disseminators of mold and mold spores. Damp cellars, summer cottages and areas with high humidity are especially susceptible to mold contamination. The concentration of fungi increases proportionately with indoor relative humidity. Year round allergy symptoms are usually caused by common molds such as Aspergillus, Pencillium, Mucor and Rhizopus. (191)

Mold spores have been identified as major causes of bronchial asthma, seasonal and perennial rhinitis (runny or stuffy nose) and eczema. Molds cause asthma symptoms similar to those caused by plant pollen, beginning with a dry non-productive cough, followed by characteristic episodes of shortness of breath and wheezing. Eye symptoms are not prominent. (189)(191).

Hypersensitivity pneumonitis is the most serious acute immune reaction, resulting in the filling and variable destruction of the lung alveoli by inflammatory cells. With continued exposure, irreversible pulmonary fibrosis occurs followed by lung failure and death. (87) Clinical symtoms of recurrent acute exposure are flu-like in nature with instances of breathlessness and non-productive cough. Continuous low level exposure is rarely manifest in constitutional symptoms before irreversible fibrosis occurs. (192) Thermophilic actinomycetes were cited as the cause of 4 out of 27 workers in one office developing hypersensitivity pneumonitis, where the organisms were dispersed by the office's air conditioning system. (193)

Humidifier fever, or humidifier lung, also involves an inflammatory reaction in the alveoli and bronchioles. It may not be easily distinguished from hypersensitivity pneumonitis since it is characterized by flu-like symptoms, including fever, chills, headaches, chest tightness and breathing difficulty. It is not known whether humidifier fever is a variant of hypersensitivity pneumonitis. No causative agent has been identified for either ailment although Thermophilic actinomycetes have been implicated by some. Table 7-3 lists various organisms isolated from faulty humidifiers which may play a causative role.

Pathogenic Viruses and Bacteria

Airborne viruses and bacteria in indoor environments are generated by the occupants. These organisms are exhaled by breathing, coughing or sneezing but may also originate in feces, urine and wounds on the skin. (84) It has been suggested however that the major threat of exposure to viruses and bacteria in indoor environments results from person to person and person to object contact rather than through inhalation of airborne organisms. (194)

TABLE 7-3
Organisms Isolated From Faulty Humidifiers

Fungi	Bacteria/actinomycetes
Alternaria spp	Flavobacterium spp
Aspergillus spp	Cytophaga spp
Aureobasidium pullulans	Alkaligenes spp
Cephalosporium spp	Bacillus spp
Chaetomium spp	Micrococcus spp
Cladosporium spp	Pseudomonas spp
Fusarium spp	Sphaerotilus
Humicola spp	*Staphylocccoccus epidermidis*
Mortierella spp	*Streptomyces albus*
Oidodendron spp	Streptomyces
Penicillium spp	*Thermoactinomyces vulgaris*
Phialophora spp	*Micropolyspora faeni*
Phoma spp	
Rhodotorula spp	Protozoa
	Acanthamoba polyphagii
	Hartmanella vermiformis
	Vahlkamfia inornata
	Oikomonas terno
	Negleria gruberi

Modified from Burge, P.S. et al. (1985) Thorax(40), 248-254.

A notable exception is the soil bacterium Legionella pneumophila, more commonly known as the causative agent of Legionnaire's disease. This disease is a multisystemic disorder which can affect the lungs, liver, kidney, gastrointestinal tract and central nervous system. Fifteen to twenty percent of those infected die from the disease, with the attack rate ranging from 0.1-5%. Person to person spreading of the disease does not appear to occur, with the only proven route of transmission being through contaminated aerosols usually originating in air handling systems. (195)

Legionnaire's disease was first reported in 1977. At an American Legion convention in Philadelphia, 182 Legionnaires contracted the disease. Twenty-

five of those cases proved to be fatal. Several outbreaks of Legionnaire's disease have since occurred. (196)

Pontiac Fever, first reported in 1968, is also caused by Legionella pneumophila bacteria. Pontiac Fever is characterized by the acute onset of fever, chills and muscle pain. Exposure often results from contaminated air conditioning systems. The incubation period for Pontiac Fever is much shorter than for Legionnaire's disease and the ailment is self-limited, usually lasting just two to seven days. (195)

MITIGATION OF BIOLOGICAL CONTAMINATION IN INDOOR ENVIRONMENTS

Adequate ventilation and overall cleanliness will help mitigate biological contaminant levels. EPA also recommends several other measures for minimizing indoor biological contamination:

1. Relative humidity levels in homes and buildings must be maintained at a level not conducive to the growth of fungi (molds and mildews), and dust mites. EPA suggests a range of 45 to 50 percent relative humidity (RH). ASHRAE recommends that humidity levels be maintained below 60% RH.

2. All places where water is likely to collect require periodic and thorough cleaning. These places include: drip pans of humidifiers and refrigerators, cooling towers, in and around toilets and wash basins, carpets and fabrics.

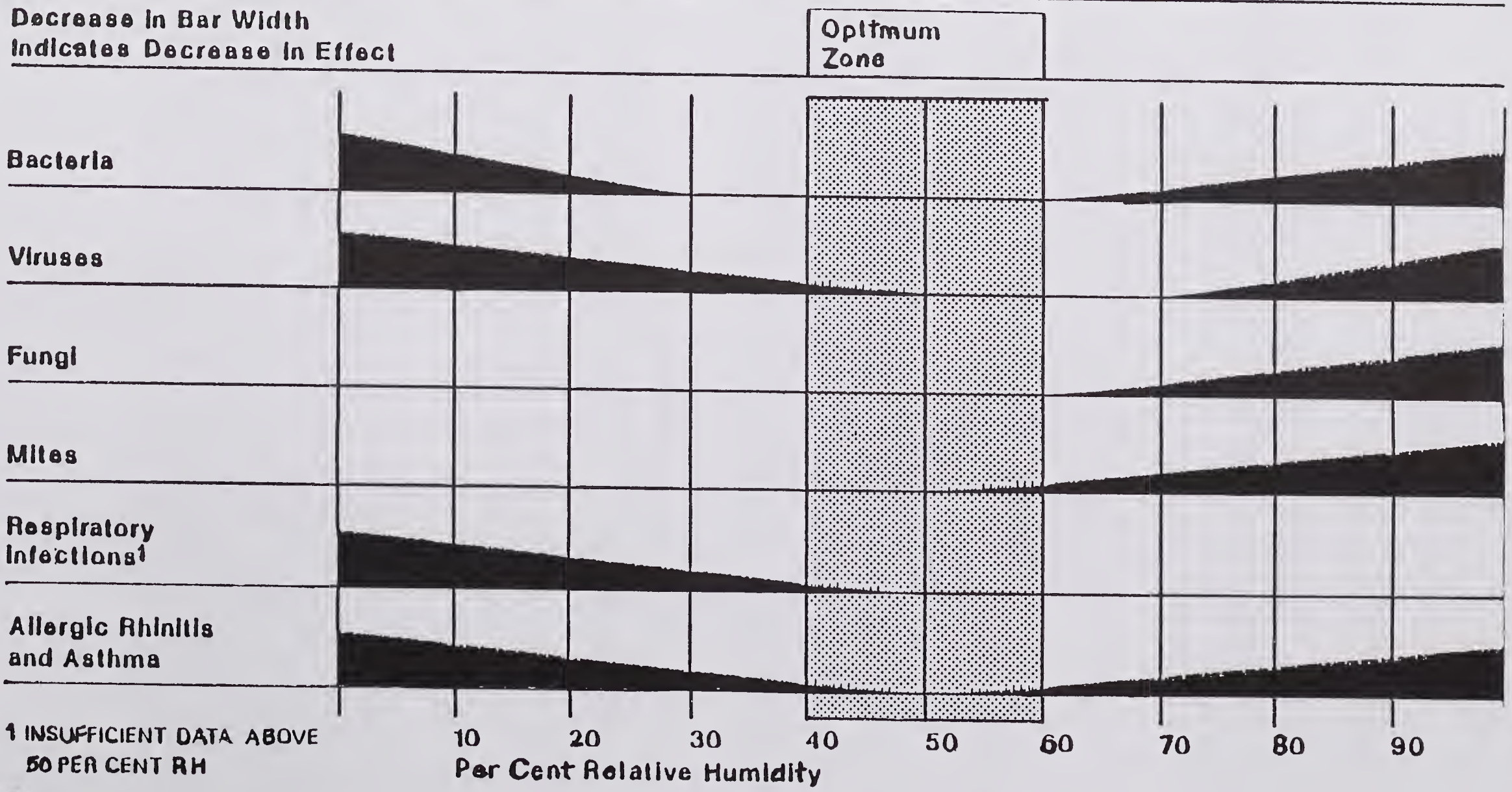
3. Household insect pests must be exterminated.

4. Periodic cleaning or replacement of furnace and air conditioning filters and other air cleaning devices must be observed. (87)

The humidity range recommended by EPA is much narrower than the current ASHRAE standard. Not only are dust mite populations eliminated, but most fungi as well. Fungi grow in the range of 75% - 95% RH. Some fungi, however, have the ability to bind atmospheric water and flourish at less than 75% RH. (197) See Figure 7-1 for the optimum RH range for various biological contaminants.

Dehumidifiers may be used to control indoor RH levels. Dehumidifying the air serves to cool it, thus obviating or at least reducing the need for air conditioning systems. Dessicant cooling represents an innovative approach to dehumidifying and thus cooling indoor air. Dessicant materials absorb moisture from the air. When the dessicant is heated, the moisture is released and vented outdoors and the dessicant is ready to absorb more moisture. Traditional air conditioners expend energy to lower air temperatures. Dessicant coolers do not. Energy is saved and refrigerants containing CFCs are not needed, while the goals of dehumidifying and cooling the air are met.

FIGURE 7-1 Optimum Relative Humidity Ranges for Health



Sterling, E.M. Criteria for Human Exposure to Humidity in Occupied Buildings. Ashrae Transactions (1985)Vol.91, Pt.1.

Humidifiers have been implicated as a major source of biological contaminants, especially cool mist humidifiers. Ultrasonic humidifiers have been touted as the solution to this problem yet recently, tests conducted in Idaho show that the use of tap water in ultrasonic humidifiers can be hazardous to health. The ultrasonic humidifier dispenses water in particles small enough to be inhaled that contain minerals present in the tap water. These minerals may have certain health effects (198).

It has been suggested that the best model for cleaning a room of microbes is a hospital operating room in which an air filter is used in conjunction with a HEPA filter, operating under positive pressure. The expense of such a system makes it implausible for residential application. (194)

Air cleaners have been described as useful in eliminating biologicals from the indoor environment. One study evaluated the ability of an electrostatic air cleaner and negative ionizer to cleanse indoor air of mold spores. Significant reductions in mold colonies were observed with the electrostatic air cleaner. No significant reduction was observed with the additional use of the negative ionizer. (199)

With the increasing use of recirculating ventilating systems, the use of high intensity UV radiation in central supply ducts may be plausible. The UV radiation would destroy many if not all biological contaminants, but would not prevent infectious person to person contacts in individual rooms. (200)

LOCAL REGULATION OF AIRBORNE BIOLOGICAL CONTAMINATION

Local boards of health have broad authority to promulgate reasonable health regulations pursuant to M.G.L. c. 111, Sec. 31 to protect the public health and safety. They have specific authority to control and regulate atmospheric pollution under M.G.L. c. 111, Sec. 31C, which may include dust and biological pollutants constituting a nuisance, a danger to the public health or an impairment to the public comfort and convenience.

Chapter 8: COMBUSTION PRODUCTS

TYPES OF COMBUSTION PRODUCTS

Complex mixtures of organic and inorganic gaseous and particulate pollutants result from the combustion of fossil fuels and vegetative materials. Fossil fuels include oil, kerosene, coal and natural gas, while vegetative materials include wood and plant material. Incomplete combustion of these fuels can contain additional types of pollutants. Table 8-1 lists sources along with gaseous and particulate combustion products. Emission rates for these pollutants are controlled by the usage pattern, burner design and manufacturer, age of burner, fuel consumption rate and combustion efficiency. Indoor concentrations of combustion products are influenced by the nature of the fuel, the location of the source, the quantity of air used, the temperature of combustion, and the presence or absence of a ventilation system. (84) Health threatening concentrations of some of these combustion products have been detected in indoor environments. Table 8-2 summarizes some known health effects of combustion products.

Carbon Monoxide

Carbon monoxide ("CO") is an odorless, colorless gas produced by the incomplete combustion of fossil fuels and wood. Gas stoves and automobiles are two significant CO sources indoors. (201) CO interferes with oxygen transport in the blood by binding to hemoglobin to form carboxyhemoglobin. Normally oxygen binds with hemoglobin to form oxyhemoglobin which is transported by the blood to tissues throughout the body, providing them with oxygen. CO binds to hemoglobin 300 times more strongly than oxygen, preventing the formation of oxyhemoglobin and thus the oxygenation of body tissues. Resultant health effects may include dizziness, blurred vision and rapid breathing. At very high doses, death may occur.

CO is emitted indoors from gas appliances, cigarettes, wood stoves, kerosene space heaters as well as cars in attached garages. Hourly concentrations of CO during cooking with a gas stove range from 2 to 6 ppm and one hour averages may exceed 12 ppm. Particular concern for CO health effects arises in the northeast where urban poor use gas stoves for heating, thus potentially increasing CO concentrations to 25 to 50 ppm. (202) Adverse health effects begin with exposure to air containing just 50 ppm carbon monoxide for ninety minutes or 15 ppm for ten hours. (84)

EPA's 8-hour air quality standard for CO is set at 9 ppm and 35 ppm for one hour, in order to prevent adverse effects in patients with cardiac and peripheral vascular disease. OSHA and ACGIH have established an occupational standard for CO of 50 ppm as an eight (8)-hour, time-weighted average; NIOSH's standard is 35 ppm as a ten (10)-hour time-weighted average.

TABLE 8-1 Indoor Air Combustion Product Sources and Related Contaminants

Sources	Gases and Vapors	Particles
Unvented appliances	H_2O, CO_2, CO, NO_x, NO_2	large particles fine particles
gas-fired		Aitken nuclei
kerosene-fired	(+)SO_2	
Ventless heaters	H_2O, CO_2, CO	
gas-fired	(+)NO_x, mercaptans	Aitken nuclei [1] fine particles
oil-fired	(+)NO_x, SO_2	large particles
wood-fired	(+)NO_x, SO_2, HC	B(a)P
Automobile garages	CO, HC, nitrogen compounds	fine particles Aitken nuclei

[1] Aitken nuclei are particles between 0.001-0.4 um in diameter upon which aerosol vapors may condense.

Woods, J.E. (1983)Sources of Indoor Air Contamination. American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc.; AC-8f-10

TABLE 8-2 Health Effects of Various Combustion Products

Contaminant	Health effect
Aldehydes	Irritation of eyes, nose, and throat
Carbon monoxide	Headaches, impairment of visual acuity and brain functioning; irregular heart functioning
Carbon dioxide	Headaches, dizziness, shortness of breath, and drowsiness
Nitric oxide	At high concentrations, irritation of eyes, nose, and throat
Nitrogen dioxide	Damage to lung tissue and increased airway resistance
Particulates	Varies according to chemical and physical properties; for example, benzo-*a*-pyrene is carcinogenic, other particulates may or may not have adverse health effects
Sulfur dioxide	Irritation of skin, eyes, and mucous membranes; at high concentration, constriction of upper airways

Turiel, I. Indoor Air Quality and Human Health, Stanford University Press, 1985.

Health effects of low level exposure have been studied but the results of these studies remain controversial. More study is justified given that CO poisoning is so well documented. In addition to the symptoms described above, neurologic manifestations of CO poisoning involve impaired mental activity, behavioral alterations and coma. Cardiac effects include arrhythmias and myocardial infarction (203).

Nitrogen Oxides

Cooking on gas stoves, burning of pilot lights and operating kerosene heaters represent some combustion processes that release nitric oxide (NO) and nitrogen dioxide (NO_2) - collectively referred to as NO_x. Continued exposure to low concentrations of NO_x may result in chronic irritation of the respiratory tract, with cough, headache, loss of appetite, dyspepsia (impaired digestion), corrosion of the teeth and gradual loss of strength. Exposure to higher concentrations may cause coughing, burning in the throat and chest, and dyspnea, possibly followed by cyanosis, loss of consciousness and death. Controlled health studies use NO_2 as an indicator for the entire scope of NO_xs.

Gas stoves appear to be the critical variable in NO_2 levels while wood fires are not strong sources. (84) Roughly 50% of homes in the U.S. have gas cooking appliances. Normal use of an unvented gas stove adds 25 ppb NO_2 to the ambient NO_2 concentration in homes. (203) Peak levels in the kitchen may reach 200 to 400 ppb. The federal standard for NO_2 in ambient air is set at just 50 ppb annual average. DEQE has established an hourly ambient guideline for NO_x of 170 ppb. These standards are applicable to outdoor environments but may be appropriate indoor guidelines.

Knowledge about NO_2 health effects is derived from animal toxicological, human clinical and epidemiological studies, most often focusing on the use of gas stoves. One NO_2 study involved the development of a model to predict personal and indoor exposure. When applied to six U.S. cities, the model showed that more than 25% of the residents of homes with gas ranges in these cities would have annual personal exposures exceeding the 50 ppb ambient standard, assuming ambient NO_2 concentrations averaged 30 ppb. (203) In addition, the urban poor who often use gas stoves for heating purposes would not only be at risk of CO poisoning as described earlier, but also for health effects resulting from exposure to NO_2.

It has been demonstrated that at typical household concentrations of 50 ppb or short periods with gas cooking or unvented space heaters, NO_2 may affect sensory perception, especially adaption to darkness, and may produce eye irritation. Several studies have shown that short-term exposure to NO levels of 1.0 to 3.0 ppm for less than one hour may increase airway resistance in humans. (204) NO_2 is an irritant to the lining of the lung which upon deposition changes chemically to form nitrous and nitric acids. (201)

Animal toxicology studies with NO_2 have shown high levels of NO_2 (greater than 2.5 ppm) causing increased susceptibility and other changes in the host's immune defenses, major structural alterations in the lung, changes in pulmo-

nary function and changes in lung biochemistry. (87) Lower levels of NO_2 (less than 1 ppm) also have been shown to cause changes in lung host defenses, lung structural changes indicative of the development of chronic lung disease, and changes in lung metabolism. These animal toxicology studies provide the basis for concern that chronic human exposure to NO_2 may result in chronic irreversible lung disease. (87)

Human clinical studies show that acute NO_2 exposure of resting, normal subjects only causes changes in pulmonary function at levels abnormally high for indoor environments, or greater than 2 ppm. (87) However, some recent studies indicate that one susceptible subpopulation may be asthmatics. Two studies showed decrements in pulmonary function in asthmatics exercising in an environment with only 0.300 ppm NO_2. (205, 206)

Most epidemiologic literature for NO_2 focuses on gas stoves. Collectively, these studies are inconclusive. Many of the studies involve school-aged children and are based on parent-completed questionnaires about the type of stove used at home. (207, 208) No consistent evidence of increased respiratory symptoms has been found in children exposed to gas stoves. Data concerning lung function in children were also inconclusive. (209, 141, 210, 211) Where some effect was shown, its magnitude was small.

Few studies examine adult health effects from indoor NO_2 exposure. As with NO_2 studies with children, no consistent adverse effects of gas stoves have been revealed relative to lung function level and chronic respiratory disease. (87)

Even with the uncertainty involved in interpreting NO_2 studies to date, available epidemiologic evidence is sufficient to assume some effect of gas stove emissions on children and possibly adults. Future studies should employ direct measurement of exposure and key in on susceptible subpopulations. Studies focusing on potential synergistic effects of NO_2 with combustion products and other indoor air pollutants should be pursued.

Respirable Particulates

Human exposure to respirable particulates resulting from combustion or cigarette smoke, such as soot or particle-bound polycyclic aromatic hydrocarbons (PAHs), most often occurs by way of the respiratory system. For the purpose of assessing the fate of particles in the respiratory system, they have been divided into fine mode particles (less than 2.5 microns) and course mode respirable particles (greater than 2.5 microns to roughly 15 microns). Fine mode and small course particles deposit in the thoracic, or tracheobronchial and pulmonary region. Course mode particles deposit in the extrathoracic, or nasopharyngeal region. Health effects resulting from deposition of particles in the respiratory tract include decreased air flow as a result of airway constriction, altered mucociliary transport, and changes in alveolar macrophage activity. (87)

Carbon Dioxide

Carbon Dioxide (CO_2) is a colorless, odorless gas formed from combustion of organic, or carbon-containing, materials and is often used as an indicator of poor ventilation. Although not normally thought of as a pollutant, unusually high concentrations of CO_2 may cause headaches, loss of judgment and asphyxiation. Outdoor concentrations typically occur between 300 to 330 ppm while the federal occupational standard is 5000 ppm and the state DPH "comfort guideline" is 600 ppm. (87) (84)

Sulfur Dioxide

Sulfur dioxide (SO_2) is generally present indoors at levels about 70% of outdoor SO_2 levels. SO_2 reacts with interior surfaces and ammonia generated by humans, thus the lower levels. Indoor values will exceed outdoor values only if kerosene is burned indoors using an unvented device with high sulfur fuel. Under these circumstances, generally only susceptible individuals such as infants or elderly people with respiratory distress are at risk. (87)

Asthmatic individuals are more sensitive to SO_2 than otherwise normal individuals. In one study, 50% of the asthmatic subjects experienced at least a doubling in airway resistance upon exposure to 0.75 ppm SO_2 or less. (212) (213)

Polycyclic Aromatic Hydrocarbons

Polycylic aromatic hydrocarbons (PAHs) are combustion products characterized by a nucleus of five or six-membered carbon rings, in which interlinked rings have at least 2 rings in common.

PAH emissions have been measured from cigarettes, furnaces, woodstoves, coal stoves, fireplaces and kerosene heaters. In one study, indoor concentrations of benzo-a-pyrene, a carcinogen, were five times higher when a wood-burning stove was in use. (214) Many PAHs are carcinogenic and may also affect the immune and cardiovascular systems.

Woodsmoke

Woodsmoke is produced by the combustion of wood and is composed of over 100 different chemicals. Several of the more significant components have been described above: CO, SO_2 and NO_x . In addition to these, many organic compounds have been identified. Table 8-3 lists many constituents of woodsmoke. The chemical and toxicological characteristics of some components may be modified significantly through chemical reactions after leaving the

TABLE 8-3 Air Pollutants in Woodsmoke

Pollutant	Carcinogenic Activity
Carbon monoxide	
Nitrogen dioxide	
Sulfur dioxide	
Acenaphthylene	
Fluorene	
Anthracene/phenanthrene	
Phenols[1]	*
Fluoranthene	*
Pyrene	*
Benz(a)anthracene	+
Chrysene	+/−
Benzofluoranthenes	+ +
Benzo(a)pyrene	+ + +
Indeno pyrene	+
Benzo(ghi)perylene	
Dibenzanthracenes	+ + +
Ancenapthene	
Ethyl benzene	
Phenathrene	
Dimethylbenzanthracene	+ + + +
Benzo(c)phenanthrene	+ + +
Methylcholanthene	+ + + +
Dibenzopyrenes	+ + +
Dibenzocarbozoles	+/− to + + +
Formaldehyde[1]	
Propionaldehyde[1]	
Acetaldehyde[1]	
Isobutyraldehyde[1]	
Cresols[1]	
Catechol	*

[1]Cilia toxic and mucous coagulating agent

+/− Uncertain or weak carcinogen; + carcinogen; + +, + + +, + + + + strong carcinogen; * initiating or cancer-promoting agents and co-carcinogenic compound

Modified from: Spengler, J.D. and Cohen, M.A., "Emissions from Indoor Combustion Sources," *Indoor Air and Human Health*, Gammage, R.B. and Kaye, S.V., eds.; and Calle, E.V. and Zeighami, Elaine A., "Health Risk Assessment of Residential Wood Combustion," *Indoor Air Quality*, Walsh, P.J., Dudney, C.S., and Copenhaver, E.D.

combustion chamber and leaking into the house through the doors of the stove or the stovepipe. Some products of such reactions have been identified as mutagens and carcinogens. (215)

Health risks associated with woodsmoke have been assessed considering the woodsmoke mixture as a whole, rather than its specific components. (215) Such an evaluation is needed since the health effects of woodsmoke may not match those of individual woodsmoke components.

Available studies on woodsmoke health effects reveal its acute and chronic effects on the respiratory system. Acute exposure may result in decrements in pulmonary function and increased incidence of respiratory symptoms. Chronic exposure to woodsmoke may play a role in the development of chronic obstructive lung disease and cancer. (215) More study is needed to correlate woodsmoke exposure to adverse health effects on a quantitative basis.

COMBUSTION PRODUCT SOURCES AND RELATED HEALTH EFFECTS

Woodstoves and Fireplaces

Even though woodburning stoves and fireplaces may be vented, woodsmoke may escape into the indoor air due to improper installation, cracks in the stovepipe, downdrafts and windows.

In Waterbury, Vermont 24 homes were tested for respirable particulate levels from woodsmoke. After seven days of sampling, it was found that homes with air-tight woodburning stoves had about 4 ug/m^3 higher indoor concentrations than those homes without woodburning stoves. (216) Another study reported indoor concentrations slightly above background levels for particulates (zero to 30 ug/m^3) with the use of air-tight stoves versus 200 to 1,900 ug/m^3 with the use of "non-airtight" stoves. (217)

Fireplaces may also cause elevated particulate levels. One study measured particulate levels inside and outside of three homes - two with fireplaces and one with a woodstove. Respirable particulate levels in each home were elevated above outdoor levels, ranging from 14.3 to 72.5 ug/m^3 in the home with the woodstove to as high as 159.9 ug/m^3 in one of the homes with a fireplace. (203) Given these findings, wood stoves and fireplaces appear to be a significant source for respirable particulates in indoor environments.

Health effects of wood combustion products have been studied infrequently. In vitro experiments have demonstrated the mutagenicity of wood smoke. (218) Few epidemiologic studies have been performed, several of which were done in less developed countries where often wood smoke exposure is much greater than in developed countries. Chronic pulmonary disease has been associated with intense woodsmoke exposure. (219) Size and density of woodsmoke particulates determine whether they will reach the lung. Those particles deposited in the lung have a greater potential to cause harm due to their longer periods of contact with the lung. Also, the chemical nature of particulates dictates in part their effect on the lungs. (84) One case in the U.S. involved an infant recurrently hospitalized for severe lower respiratory tract disease. The disease ceased following removal of a wood stove from the infant's home. (220)

Benzo-a-pyrene is an organic carcinogen bound to particulates resulting from the combustion of both wood and animal dung. Benzo-a-pyrene may inter-

fere with the clearance mechanisms of the respiratory tract. As mentioned earlier, woodsmoke also contains other hydrocarbons which will be discussed below along with other types of volatile organic compounds.

Federal standards of performance have been established for particulate emissions from new residential wood heaters. The need for these regulations resulted primarily from the drastic increase in residential woodstove use. Currently, there are about twelve million wood stoves in use with more than a half million units added each year. (221)

Woodstoves with catalytic combusters shall not discharge into the atmosphere any gases which contain particulate matter in excess of a weighted average of 5.5 g/hr., or 8.5g/hr for woodstoves without catalytic combusters. These standards apply only to woodstoves manufactured on or after July 1, 1988 and sold at retail on or after July 1, 1990. A stricter standard applies to woodstoves manufactured on or after July 1, 1990 or sold at retail on or after July 1, 1992; that is, woodstoves with a catalytic combuster must not exceed 4.1 g/hr. and woodstoves without a catalytic combuster must not exceed 7.5 g/hr.

Kerosene Heaters

Unvented kerosene heaters emit a range of pollutants including NO_x, CO, CO_2 , SO_2 and formaldehyde. Such unvented kerosene heaters vary in their output of pollutants due to wick height, fuel type, adjustment of primary air/fuel ratio, the length of time the burner is used over a given period and the overall design of the heater. (222)

Studies specific to kerosene heaters have been conducted to identify and quantify pollutants emitted. The Oak Ridge National Laboratory (ORNL) has collected preliminary data from homes in which kerosene heaters were used for more than 100 hours per week. Passive monitors detected NO_2 levels ranging from .01 ppm to .15 ppm.

Average concentrations when heaters were in use ranged from 0 to .56 ppm, while NO_2 concentrations in homes without kerosene heaters in use averaged .007 ppm. These NO_2 levels in homes using kerosene heaters are in the range where previous studies have shown some level of NO_2 health effects. (223) As a result, CPSC is working to establish an NO_2 emission rate from kerosene heaters, not to exceed .3 ppm.

In addition to those listed above, kerosene heaters emit aliphatic hydrocarbons, alcohols, ketones, phthalates, and alkyl benzenes. (224) These volatile organic compounds will be discussed collectively below.

Gas Stoves

Gas stoves are used in almost half of the U.S. homes and most do not vent combustion products. Gas stoves are a source for particulates, NO_x, SO_2, formaldehyde, and CO. Newer stoves, while they have a new burner design, do not consistently show higher or lower emissions than older stoves. (87)

Roughly 30% of NO_2 and CO emissions are emitted from the pilot lights still on 80-90% of gas stoves in use in Massachusetts. Massachusetts currently prohibits the use of pilot lights on new gas stoves as do a number of other states.

Gas-fired Space Heaters

Unvented gas fired space heaters emit NO_x, CO, CO_2 , respirable particulates and formaldehyde. Factors affecting emissions are usage patterns, brand of heater, burner design, size of heater and the tuning of the fuel to air mixture. (225)

Tests of gas-fired infrared heaters versus blue flame heaters revealed average NO_2 emissions from the infrared heater to be one fourth to one-half the NO_2 emissions from the blue flame heaters. (226) Propane fired heaters exhibited the highest NO_2emissions, followed by blue flame heaters with about one half the NO_2emissions of propane heaters. Virtually no NO_2emissions were detected from the infrared heaters.

Relative to CO, the infrared heaters had the highest emissions, followed by the blue flame and finally propane fired heaters. Propane heaters exhibited virtually no unburned hydrocarbon emissions as opposed to blue flame and infrared heaters.

Gas-fired Water Heaters, Furnaces and Dryers

Faulty venting systems in gas-fired water heaters, furnaces and dryers have resulted in elevated indoor concentrations of NO_2. Corrosion in gas furnaces could allow dangerous CO to escape into home living areas. Testing has shown furnaces to be susceptible to corrosion when exposed to chlorine- or fluorine-containing compounds such as salt, paint stripper, fabric or water softeners, bleach and adhesives. (227)

In Virginia, a lawsuit was filed against the owners of an apartment in which an improperly maintained furnace and hot water heater emitted CO into the apartment. One 28 year old occupant of the apartment died of carbon monoxide poisoning. (227)

MASSACHUSETTS REGULATION OF INDOOR COMBUSTION APPLIANCES

Space Heaters and Portable Stoves

Massachusetts laws regulate several types of space heaters and portable stoves. The following space heaters are prohibited in buildings inhabited by people: wick-type space heaters (MGL c. 148, Sec. 5A), those burning kerosene, range oil, or number 1 fuel oil (MGL c. 148, Sec. 25B), and unvented gas or oil space heaters (MGL c. 148 Sec. 25A). In addition, the sale or installation of secondhand space heaters or portable stoves burning kerosene, range oil or number one fuel oil is prohibited (MGL c. 148, Sec. 25A).

Gas Appliances

Massachusetts laws also regulate gas applicances. For example, gas appliances (manufactured after June 1, 1988) sold or installed in this state must employ intermittent ignition devices (MGL c. 148, Sec. 25E), no pilot lights are permitted. In this context, "gas appliances" include heaters, stoves, clothes dryers, etc., but exclude water heaters for domestic use, kitchen heating ranges, and space heaters. "Intermittent ignition devices" activate only when the gas unit is ignited prior to use.

Local Control of Combustion Pollution

Local boards of health have general authority under M.G.L. c. 111, Sec. 31C to regulate and control atmospheric pollution. Such pollution includes the emission of smoke, particulate matter, soot, cinders, ashes, vapors and odors, all of which originate from indoor combustion sources. These pollutants may be regulated if they constitute a nuisance, a danger to the public health or an impairment to the public comfort and convenience.

Chapter 9: VOLATILE ORGANIC COMPOUNDS

WHAT ARE VOLATILE ORGANIC COMPOUNDS?

Volatile organic compounds (VOCs) are the large category of carbon-based compounds that evaporate. One of the most ubiquitous and hazardous VOCs present in indoor environments is formaldehyde, discussed in detail in Chapter 4. Other types of VOCs including their sources and health effects are summarized in Table 9-1. VOCs are present in gaseous form and therefore can easily bypass respiratory clearance mechanisms and those VOCs that are water-soluble can even dissolve in the bloodstream. Health effects range from irritating effects to neurotoxic and carcinogenic effects.

SOURCES OF VOLATILE ORGANIC COMPOUNDS

As many as 300 organic compounds have been identified in homes in one of the U.S. EPA's Total Exposure Assessment Methodology (TEAM) studies (10). Various other studies have revealed in excess of 250 different VOCs present in indoor environments at levels greater than 1 ppb. (228) VOCs have a wide variety of sources: combustion of organic materials, emissions from building materials, furnishings, office equipment and supplies, emissions from pesticide products and tobacco smoking and many common consumer products, including household cleaners, air fresheners, hair sprays, dry-cleaned clothing and chemicals used for hobbies, such as furniture refinishing. A recent EPA study identified major indoor contributors of many VOCs to be adhesives and paint while foam insulation, vinyl and rubber molding, particle board, and telephone cables are major sources for individual VOCs. (229) The authors further concluded that elevated VOC levels in new and renovated buildings clearly may cause Sick Building Syndrome ("SBS") symptoms.

VOCs are also adsorbed by building material surfaces and subsequently reemitted into the indoor air. It has been shown that the amount of VOC adsorbed and reemitted depends upon the total surface area exposed. Also, the lower the ventilation rate, the more VOCs will be adsorbed on these interior surfaces. (230) See Table 9-2 which identifies common VOC sources in homes.

EPA'S TEAM STUDY

In one TEAM Study spanning from 1979 to 1985, the main goals were to (1) develop methods for measuring individual exposure and resultant body burden of toxic and carcinogenic chemicals, and (2) to apply these methods to estimate exposure and body burdens of urban populations in several U.S. cities.

TABLE 9-1 Selected Organic Compounds and Their Health Effects

Compound	Health effects	Sources and uses
Formaldehyde and other aldehydes	Eye and respiratory irritation; possibly more-serious long-term health effects	Outgassing from building materials (particle board, plywood, and urea-formaldehyde insulation foam); also from cooking and smoking
Benzene	Respiratory irritation; recognized carcinogen	Plastic and rubber solvents; from cigarette smoking; in paints and varnishes, including putty, filler, stains, and finishes
Xylene	Narcotic; irritating; in high concentrations, possibly injurious to heart, liver, kidney, and nervous system	Solvent for resins, enamels, etc.; in non-lead automobile fuels and in manufacture of pesticides, dyes, pharmaceuticals
Toluene	Narcotic; may cause anemia	Solvents; by-product of organic compounds used in several household products
Styrene	Narcotic; can cause headache, fatigue, stupor, depression, incoordination, and possible eye injury	Widespread in manufacture of plastics, synthetic rubber, and resins
Trichloroethane	Subject of OSHA carcinogenesis inquiry	Aerosol propellant, pesticide, cleaning solvents
Trichloroethylene	Animal carcinogen; subject of OSHA carcinogenesis inquiry	Oil and wax solvents, cleaning compounds, vapor degreasing products, dry-cleaning operations; also as an anesthetic
Ethyl benzene	Severe irritation to eyes and respiratory system	Solvents; in styrene-related products
Chloro benzenes	Strong narcotic; possible lung, liver, and kidney damage	In production of paint, varnish, pesticides, and various organic solvents
Polychlorinated biphenyls (PCB's)	Suspected carcinogens	In various electrical components; in waste oil supplies and in plastic and paper products in which PCB's are used as plasticizers
Pesticides	Suspected carcinogens	Insect control

SOURCE: C. D. Hollowell and R. R. Miksch, *Sources and Concentrations of Organic Compounds in Indoor Environments*, Lawrence Berkeley Laboratory Report LBL-13195 (July 1981).

TABLE 9-2 SPECIFIC INDOOR SOURCES OF ORGANIC VAPORS

Compound	Material Source(s)	Reference
Paradichlorobenzene	Moth crystals, Room deodorants	Nelms et al. (1987)
Methylene Chloride	Paint removers	Girman and Hodgson (1986)
Chloroform	Chlorinated water	Wallace (1986b)
Formaldehyde	Pressed wood products, Foam insulation (UFFI), Textiles, Disinfectants	National Research Council (1981)
Styrene	Plastics, Paints	Wallace (1986b)
Toluene Diisocyanate	Polyurethane foam aerosols	Carroll et al. (1976)
Phthalic Acid Anhydride, Trimellitic Acid, Triethylene Tetraamine	Epoxy resins	Fawcett et al. (1977)
Sodium Dodecyl Sulfate	Carpet shampoo	Kreiss et al. (1982)
Benzyl Chloride, Benzal Chloride	Vinyl tiles plasticized with butyl benzyl phthlate	Rittfeldt et al. (1984)
Ethylene Oxide	Sterilizers (Hospitals)	

Source: U.S. E.P.A. Preliminary Indoor Air Pollution Assessment- Appendix A

Residents from New Jersey, North Carolina, North Dakota and California carried air samplers and also gave a daily breath sample. Personal exposures to VOCs were consistently higher than outdoor concentrations with indoor VOC sources appearing to be largely responsible for this finding. Breath concentrations of VOCs also exceeded outdoor concentrations and correlated more strongly with personal exposure than outdoor concentrations. (10) A recent study conducted in the Kanawha Valley of West Virginia corroborated this finding of higher VOC levels indoors than outdoors (231). The results of this TEAM Study clearly demonstrate the prominant role indoor sources of VOCs play in human exposure. See Table 9-3 for typical sources of residential VOC exposure.

THE FEDERAL INTERAGENCY INTEGRATED CHLORINATED SOLVENTS PROJECT

The Interagency Integrated Chlorinated Solvents Project is chaired by EPA's Office of Toxic Substances. Other EPA offices are involved as well as the Consumer Product Safety Commission (CPSC), the Occupational Safety and Health Administration (OSHA), and the Food and Drug Administration (FDA). The project is considering risks associated with specific chlorinated solvents: methylene chloride, perchloroethylene, trichloroethylene, 1,1,1-trichloroethane, CFC-113, and carbon tetrachloride. The project's purpose is to determine appropriate control and regulatory options for these substances.

One of the VOCs under study, perchloroethylene (perc), is a dry cleaning solvent which presents an indoor pollution hazard in both the homes of consumers and in industrial work environments like dry cleaners and metal degreasing shops. Some studies have found that dry cleaning employees suffer from increased urinary tract cancers. Perc cannot be confirmed as the cause since dry cleaners used hydrocarbon solvents for years before switching to perc. (232)

Methylene chloride is a solvent used in paint strippers, spray paints, cleaning fluids and other products. The Consumer Federation of America requested the CPSC to ban methylene chloride in 1985. Instead, CPSC instituted a three-stage plan to evaluate whether consumer exposure to methylene chloride is being reduced as a result of CPSC's mandatory labeling requirements for consumer products containing the substance, designation of methylene chloride as hazardous and initiation of public information compaigns. (233)

TABLE 9-3 Probability of Exposure to Volatile Organics

Activities, occupations, or household characteristics associated with significantly increased exposures in air or exhaled breath to eleven prevalent chemicals in New Jersey.

Chemical	Rank	Activity[a]	p[b]
Benzene	1	Smoking	0.00001
	2	Having a smoker in the home	0.0006
	3	Being exposed to smokers	0.02
	4	Visiting a dry cleaners	0.03
	5	Traveling in a car	0.04
Styrene	1	Smoking	0.00001
	2	Having a smoker in the home	0.0001
	3	Working at a plastics plant	0.0005
	4	Exposed to paints	0.002
	5	Working at/being in a paint store	0.005
	6	Working at a chemical plant	0.007
	7	Building scale models	0.007
	8	Painting as a hobby	0.009
	9	Being nonwhite	0.01
	10	Metalworking	0.02
	11	Working with degreasers	0.03
Ethylbenzene	1	Smoking	0.0001
	2	Exposed to high dust/particle levels	0.0002
	3	Having a smoker in the home	0.0006
	4	Working with solvents	0.001
	5	Wood processing	0.001
	6	Working at a service station	0.002
	7	Having a chemical workers in the home	0.002
	8	Employed	0.002
	9	Living in a home < 1 yr	0.003
	10	Pumping gas	0.005
	11	Metalworking	0.005
	12	Working at a scientific lab	0.005
	13	Refinishing furniture as a hobby	0.01
	14	Working with dyes	0.01
	15	Having a metal worker in the home	0.02
	16	Working with odorous chemicals	0.02
	17	Having a furniture refinisher in the home	0.04
m, p-Xylene	1	Employed	0.0001
	2	Smoking	0.0001
	3	Wood processing	0.0001
	4	Working at a service station	0.0001
Carbon tetrachloride	1	Aged less than 17	0.0005
	2	Metalworking	0.006
	3	Working at/being in a paint store	0.02
	4	Furniture refinishing	0.03
m, p-Dichlorobenzene	1	Working at a hospital	0.0001
	2	Having central air conditioning	0.004
	3	Treating home with pesticides	0.05
Chloroform	1	Working at/being in a paint store	0.007
	2	Using pesticides	0.02

TABLE 9-3 (cont'd)

	5	Having a chemical worker in the home	0.0001
	6	Working with solvents	0.0003
	7	Having a smoker in the home	0.0006
	8	Living in an old home (>10 yrs)	0.002
	9	Living in a new home (<1 yr)	0.003
	10	Pumping gas	0.006
	11	Metalworking	0.008
	12	Exposed to high dust/particle levels	0.01
	13	Having a furniture refinisher in the home	0.02
	14	Furniture refinishing	0.03
o-Xylene	1	Wood processing	0.0001
	2	Employed	0.0001
	3	Working with solvents	0.0008
	4	Working with odorous chemicals	0.001
	5	Pumping gas	0.002
	6	Metalworking	0.003
	7	Having a chemical worker in the home	0.003
	8	Having a smoker in the home	0.005
	9	High dust/particle levels	0.006
	10	Having a furniture refinisher in the home	0.006
	11	Living in an old home (>10 yrs)	0.007
	12	Furniture refinishing	0.02
	13	Aged between 40 and 65	0.03
1,1,1-Trichloroethane	1	Wood processing	0.0001
	2	Employed	0.0001
	3	Working at a textile plant	0.007
	4	Metalworking	0.006
	5	Having a metal worker in the home	0.008
	6	Having a chemical worker in the home	0.009
Trichloroethylene	1	Wood processing	0.002
	2	Working at a plastics plant	0.003
	3	Gas furnace	0.01
	4	Working at a scientific lab	0.01
	5	Smoking	0.02
Tetrachloroethylene	1	Employed	0.0001
	2	Wood processing	0.0002
	3	Visiting a dry cleaners	0.003
	4	Working at a textile plant	0.01
	5	Using pesticides	0.01
	6	Working at/being in a paint store	0.03
	7	Being male	0.04

[a]Based on questionnaire data from 350 subjects in Bayonne-Elizabeth, NJ Fall 1981

[b]Probability that the association is due to chance

Wallace,L.A. Personal Exposures, Indoor and Outdoor Air Concentrations, and Exhaled Breath Concentrations of Selected Volatile Organics Compounds Measured for 600 Residents of New Jersey, North Dakota, North Carolina, and California. Toxicology and Environmental Chemistry, 1987.

STATE INITIATIVES

DEQE's Division of Air Quality Control (DAQC) is investigating controls for small but ubiquitous sources of VOC emissions due to their contribution to unhealthful ozone levels.

Movement to control VOC emissions from consumer products has also been initiated. The Northeast States for Coordinated Air Use Management (NESCAUM), of which the DAQC is a member, plans to release a study in July 1989 on VOC emissions from consumer products and how these emissions can be controlled. At least three (3) other states are developing strategies for controlling VOC emissions from consumer products including California, New York, and New Jersey.

The DAQC has participated in NESCAUM's research on the health effects of two specific VOC's sometimes present indoors: trichloroethylene and perchloroethylene. Regulations to control various VOC emissions are being considered by DAQC now. Control of architectural coating alone has been projected to reduce the state's total VOC emission inventory by 1%, or 2,200 tons annually. Control of emissions from air fresheners, hair care and personal hygiene products and disinfectants is also being considered.

LOCAL VOC CONTROL

Under M.G.L. c. 111, Sec. 31C, Massachusetts boards of health have authority to control and regulate atmospheric pollution, including the emission of fumes, vapors and gases, i.e. VOCs tat constitute a nuisance, a danger to the public health or an impairment to the public comfort and convenience. The Commission knows of no currently effective VOC-specific local health regulations.

VOC HEALTH EFFECTS

Some individual VOCs found indoors are known to be neurotoxic, such as n-hexane, MEK, and toluene. The neurotoxic effects of these VOCs individually have not been seen at levels as low as those generally found indoors. Neurotoxic symptoms of many VOCs include central nervous system depression, unconsciousness, vertigo and visual disorders. Other effects less often associated with VOCs are tremor, fatigue, anorexia, weakness as well as cognitive effects such as memory impairment and mental confusion. (87)

EPA employees recently fell victim to SBS following renovation of their Washington offices. Employees complained of dizziness, headaches, rashes, breathing difficulties, nausea and fatigue. The suspected cause is a VOC -

4-phenylcyclohexane ("4-PC") along with an inadquate ventilation system. Currently available scientific evidence links 4-PC with such adverse health effects as skin, eye and respiratory irritation. (234)

It has been suggested and the EPA case as well as numerous other such cases exemplify that the sensory irritant symptoms associated with SBS may be caused by additive or synergistic VOC activity. (87) Even though the current literature does not indicate VOC health effects at levels found indoors, literature concerning such potential additive or synergistic effects is negligible. Therefore, the pattern of VOC health effects elucidated from various studies to date warrants further study to determine VOC exposure levels at which health effects occur and whether VOCs act synergistically or additively to cause SBS. (235)

MITIGATION OF INDOOR VOC CONTAMINATION

Ventilation

Increasing air exchange rates is the most commonly employed means for reducing VOC concentrations by dilution and flushing. Care in designing changes to ventilation systems is needed, especially since in some cases, increasing the air exchange rate may increase the VOC emission rate by enhancing VOC evaporation rate. (87)

Adsorption Air Cleaners

Air cleaners may be designed for large industrial or commercial buildings or for single residential rooms. Virtually no data exist on the efficiency of air cleaning devices below pollutant concentrations of 100 ppm. (87) The rate of VOC reduction with increased ventilation may vary with different VOC contaminants.

Catalytic Oxidation Air Cleaners

More study is needed to assess the effectiveness of catalytic oxidation in removing organic vapors. The device has been shown to remove a variety of indoor contaminants resulting from combustion processes, tobacco smoking, acetone and methylene chloride. Benzene is not removed. (87)

Material/Product Selection

Careful selection of building materials, furnishings and household cleaning products would drastically reduce indoor levels of VOCs. One study noted higher concentrations of p-dichlorobenzene, decane, methyl chloroform and trichloroethylene in homes with mothballs or air fresheners than in homes without these substances (231). The same study found higher concentrations of benzene, ethyl benzene, m-xylene, o-xylene, trimethylbenzene, carbon tetrachloride, ethylene dichloride, methyl chloroform, trichloroethylene, chlorobenzene, p-dichlorobenzene and decane in homes with attached garages than in houses without attached garages (231).

Some experiments have been completed showing that at a total VOC concentration of 5000 mg/m^3, irritation effects begin. As a result, it has been suggested that material sources emitting more than 100 mg/hr of VOCs per 100 square meters of residential space should be avoided. Additional characterization of such indoor products is needed to make these product selection determinations. (87)

Material/Product Use

Some VOC emissions from products decline over time, so aging of these products before use would mitigate some VOC contamination. In California, a "bake-out" process has been used where newly built or renovated buildings are heated and ventilated prior to being occupied. The purpose of such a "bake-out" in theory is to increase the emissions of VOCs (the greater the temperature and the greater the ventilation rate, the greater the emission of VOCs). At this point, very little is known about what "bake-out" protocol is best and whether any undesirable side effects may occur. At least in the short-term following a "bake-out," VOC levels have been shown to decrease by 29% from pre- "bake-out" levels. It is unknown whether this decrease is permanent or transitory and whether it is simply a result of the normal aging that would have ocurred anyway in the time between VOC measurements. (236)

Chapter 10: PESTICIDES

OVERVIEW OF PESTICIDES

Pesticides control unwanted insects, fungi, plants or rodents, in the form of insecticides, fungicides, herbicides, or rodenticides. Because pesticides are formulated to be toxic, they can pose a sizable threat to exposed humans. Consequently, it is imperative that pesticides be thoroughly assessed for human toxicity, and the most toxic avoided. Application procedures should also be designed to minimize human exposures.

Several characteristics peculiar to pesticides increase their risk to humans. For example, in addition to exposure from the initial application, exposure may continue to occur much later since pesticides are frequently designed to be long-lasting. The likelihood of human exposure is greatly enhanced with such pesticides. Chlordane, for example, a termiticide whose production is being discontinued, lingers in soil or wood up to five years. The ability of some pesticides to bioaccumulate in the human body must be considered as well. Many pesticides are fat soluble and may build up in adipose (fat) tissue in the body. Chronic, low-level exposures could eventually prove dangerous. In sum, pesticides should be recognized as hazardous compounds and should be used minimally and selectively with caution.

ACTIVE AND INERT INGREDIENTS

Ingredients in pesticides are classified as active or inert. Active ingredients perform the pesticidal actions, whereas inert ingredients such as fillers, binders and solvents merely facilitate the active ingredients. As noted by Jeff Carlson of the Massachusetts Department of Food and Agriculture's Pesticide Bureau, most of the thousands of U.S. pesticides are formed from a combination of 1,800 approved active ingredients. Only occasionally are new active ingredients registered. (237)

Ingredients are registered pursuant to the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA). Before a new pesticide can be registered, it must undergo testing for efficacy as a pesticide as well as testing for toxicity. Finally, after field testing, the EPA determines if the product should be registered.

Unfortunately, many pesticides were registered before these measures of evaluation were adopted. 1988 FIFRA amendments, signed into law October 1988 (Public Law 100-532), require EPA to re-analyze approximately 1400 active ingredients, grouped into about 600 classes, for health and environmental effects using today's criteria. (238) 1978 FIFRA amendments had initiated this re-evaluation. However, the General Accounting Office determined that

this 1978-enacted study would not be completed until 2024, due to inadequate funding. (239) If funding for the 1988 Amendments remains intact, reassessment of these 600 classes of active ingredients should be completed in nine years. In any event, the re-registration process has been subject to severe delays and as a result pesticides with potentially serious adverse effects remain on the market.

Until recently, little attention has centered on inert ingredients in pesticides. In fact, inert ingredients are rarely noted on pesticide labels. In 1987 the EPA began to study some inert ingredients, classifying them into lists. Since then, the lists have changed slightly and updated versions will be published in mid-1989 according to EPA's Pesticides and Toxic Substances Registration Division (240). In 1989, List 1 included fifty-six compounds classified as toxic. Some of these include asbestos fibers, benzene, cadmium compounds, and carbon tetrachloride. Formaldehyde, from List 1, has been reclassified as an active ingredient. List 2 contains some sixty compounds which EPA considers potentially toxic and plans to test. Toluene, cyclohexanone, and cresols are some inert ingredients from List 2. List 3 contains approximately 800 ingredients of unknown toxicity and, List 4 consists of about 300 which are considered harmless. The EPA has stated it does not plan to regulate compounds from List 3 and List 4 at this time.

The EPA plans to discourage industry from using List 1 inert ingredients. As a primary deterrent, ingredients from List 1 are or will be subject to data call-ins. EPA has the authority to require data call-ins, for which pesticide producers must submit toxicology data on compounds of reasonable concern. EPA's first notice for List 1 data call-ins was in April 1988. The final notice, incorporating the remainder of the List 1 ingredients, will probably be in March or April 1989. Because these data call-ins frequently entail extensive and expensive studies, pesticide producers may prefer instead to utilize less toxic inert ingredients in formulating their pesticides. Should a producer continue to use List 1 compounds, they must now affix warning labels on their pesticides. This may also have a deterrent effect. (240)

MODE OF EXPOSURE

Pesticides can be absorbed through the skin, ingested through the mouth, or inhaled into the lungs. When pesticides are tested, all three modes of exposure are evaluated. The EPA determines dosages necessary to kill half of test animal populations via the three routes of exposure. LD 50 is the experimentally determined lethal dose for half of the test population from skin and oral exposures; LC 50 is the lethal dose for half from inhalation. (LD units are milligrams of pesticide per kilogram animal weight and LC units are milligrams of pesticide per liter of air.) The lower the value of LD 50 or LC 50, the more potent the poison.

Chronic effects are also studied in animal popoulations. Pesticides are tested for neurotoxicity, carcinogenicity, mutagenicity (causing gene muta-

tions), teratogenicity (causing fetus deformities), and oncogenicity (inducing tumors). (241)

The EPA began a Non-occupational Pesticides Exposure Study (NOPES) in 1985, attempting to evaluate pesticide exposure from air, drinking water and skin contact in homes located in two American cities. Because of insufficient data, dermal exposures could not be determined. However, five pesticides - chlorpyrifos, diazinon, chlordane, propoxur, and heptachlor - were detected in the air of 80-100% of the pilot homes tested. With one exception, air concentrations did not exceed acceptable levels. In contrast, no pesticides were found in tap water in any homes. Although the findings are preliminary, this study suggests that inhalation of pesticides, rather than ingestion, may be a primary pathway for exposure to pesticides indoors. (242)

With respect to indoor concentrations of pesticides, a recent study found indoor levels of four pesticides - heptachlor, chlorpyrifos, gamma- and alpha-chlordane, and trans-nonachlor - to exceed outdoor levels. (243) As with many environmental pollutants, indoor exposures may prove more hazardous than outdoor exposures.

SOURCES OF INDOOR PESTICIDE EXPOSURE

Roughly 90% of pesticide usage is for agricultural purposes. (87) Table 10-1 demonstrates that numerous pesticide sources exist leading to exposures in indoor environments. The U.S. General Accounting Office (GAO) ranked fifty common pesticides for their prevalence of use in nonagricultural settings such as homes, gardens, stores, restaurants, offices and golf courses. (244) Much of the data from this 1986 report was obtained from a 1984 EPA National Urban Pesticide Applicators Survey (NUPAS). (See Table 10-2) (245) Taken together, Table 10-1 and 10-2 provide a comprehensive compilation of pesticides which could likely contaminate indoor environments.

In addition to this national ranking, the GAO report provided three charts enumerating insecticide, herbicide and rodenticide use in various Boston facilities or locations. (See Tables 10-3, 10-4 and 10-5). (245)

Some examples of the more prevalent pesticides contaminating indoor environments are chlordane, chlorpyrifos and sulfuryl fluoride. Chlordane was used as a termiticide for decades for a number of reasons. It is effective, inexpensive, long-lasting, and has low volatility. However, it was banned by the EPA in 1974 except for subterranean termiticide use. Chlordane is now banned in Massachusetts, and is being phased out nationwide. Because it may persist for years, as noted earlier, exposures continue to be of concern. Interestingly, boric acid, a relatively non-toxic, effective termiticide, is ranked fifty-nine in use by NUPAS.

TABLE 10-1 (continued)

Compound (Trade Name)	Type of Pesticide	Household Uses Leading to Potential Human Exposure
2,4-D esters	Herbicide	Post-emergent weed control
Malathion	Insecticide	Insect control on fruits, vegetables, ornamentals, and inside homes
Permethrin (cis and trans)	Insecticide	Control of flies, mosquitoes, ants, cockroaches, garden insects
Heptachlor	Insecticide	Subterranean termite control
Aldrin	Insecticide	Subterranean termite control
Dieldrin	Insecticide	Subterranean termite control
Ronnel	Insecticide	Fly and cockroach control
Diazinon	Insecticide, Nematicide	Control of soil and household insects, grubs and nematodes in turf; seed treatment and fly control
Methoxychlor	Insecticide	Control of insects in garden, fruit, and shade trees
Atrazine	Herbicide	Weed control
α-Hexachlorocyclohexane (α-BHC)	Insecticide	Manufacture and use discontinued in U.S.; ubiquitous in air, residue from lindane
Bendiocarb (Ficam®)	Insecticide	Household, ornamental, and turf insect control
Folpet	Fungicide	Fungus control on flowers, ornamentals, seeds, plant beds; paints and plastics
Chlorothalonil (Bravo©)	Fungicide	Broad spectrum fungicide; wood preservative; paint additive
Dacthal	Herbicide	Selective pre-emergent weed control on turf, ornamentals, and vegetable crops

(continued on following page)

TABLE 10-1 SOURCE OF INDOOR PESTICIDE EXPOSURE

Compound (Trade Name)	Type of Pesticide	Household Uses Leading to Potential Human Exposure
Chlorpyrifos (Dursban®)	Insecticide	Control of mosquitoes, cockroaches and other household insects; turf and ornamental insects; fire ants, termites, and lice
Pentachlorophenol	Fungicide Insecticide	Exterior wood preservative
Chlordane	Insecticide	Subterranean termite control
Ortho-Phenylphenol	Disinfectant, Fungicide	Household disinfectant; post-harvest application to fruits and vegetables
Propoxur (Baygon®)	Insecticide	Control of cockroaches, flies, mosquitoes; lawn and turf insects
Resmethrin	Insecticide	Control of flying and and crawling insects; fabric protection; pet sprays and shampoos; application on horses and in horse stables; greenhouse use
Dicofol	Insecticide	Control of mites on fruit, vegetable, and ornamental crops
Captan	Fungicide	Seed protectant; fungal control on fruits, vegetables, and berries
Carbaryl (Sevin®)	Insecticide	Control of insects on lawns, ornamentals, shade trees, vegetables, and pets
Lindane (γ-BHC)	Insecticide	Seed treatment; insect control in soil, on vegetables, ornamentals and fruit and nut trees
Dichlorvos (DDVP)	Insecticide	Household and public health insect control; flea collars and no-pest strips

(continued on following page)

TABLE 10-1 (continued)

Compound	Type of Pesticide	Household Uses Leading to Potential Human Exposure
Oxychlordane	--	Oxidation product of chlordane
Heptachlor epoxide	--	Oxidation product of heptachlor
trans-Nonachlor	--	Component of chlordane
PCBs (Aroclors 1242 and 1260)	--	Used in electrical transformers until 1976

Source: Lewis et al. (1986).

Sulfuryl fluoride is a fumigant, a gaseous pesticide. EPA characterizes fumigants with "extraordinary power to penetrate the lining membranes of the respiratory and gastrointestinal tracts, and the skin." (244) Of the various types of pesticides, fumigants in particular have the greatest potential for compromising indoor air quality.

Chlorpyrifos is considered "moderately toxic" by EPA. (See Table 10-3), (244) Its health effects are thought to be chiefly neurotoxic. Chlorpyrifos has been postulated as a primary source for building illnesses. (245)

FEDERAL REGULATION OF PESTICIDES

Under the Federal Insecticide, Fungicide and Rodenticide Act (FIFRA), and its regulations (40 CFR 158), manufacturers, processers, distributors and retailers of pesticides must register them with the U.S. EPA. FIFRA was first passed in 1947. It was later amended in 1972 to require additional evaluation of pesticide toxicity to be submitted by the registrant. Pesticides are registered for general use or for restricted use only by a certified applicator. The U.S. Food and Drug Administration regulates pesticide residues on foods. To date, roughly 40,000 pesticides have been registered in the United States. (246)

TABLE 10-2

Sample of 50 Chemicals Used in Nonagricultural Pesticide Products

Chemical	Type of pesticide[a]	NUPAS ranking	Top 10 homeowner chemical?
2,4-D[b]	H	none[b]	Yes
Chlordane	I	2	Yes
Sulfuryl Fluoride	I	6	
Diazinon	I	7	Yes
Chlorpyrifos (Dursban)	I	9	Yes
Betasan	H	11	
Heptachlor	I	13	
Atrazine	H	14	
Dacthal (DCPA)	H	16	
Carbaryl	I	17	Yes
Methoxychlor	I	18	Yes
Aldrin	I	20	
Malathion	I	21	Yes
Diuron	H	22	
Bromacil (Hyvar X)	H	23	
Sodium Metaborate	H	24	
Sodium Chlorate	H	25	
Dichlorvos (DDVP)	I	28	
Simazine	H	31	Yes
Bendiocarb	I	32	
Parathion	I	33	
Dimethylamine Dicamba	H	34	
Metolachlor	H	36	
Dicofol	I	37	
Prometon	H	38	
Alachlor	H	39	
Polybutene	R	40	
Trichlorfon	I	42	
Toxaphene	I	44	
Endothall, Dipotassium salt of	H	47	
Aspon	I	48	
Diquat Dibromide	H	49	
Benefin	H	50	
Piperonyl Butoxide	I	51	
Glyphosate	H	52	
Lindane	I	53	
Acephate	I	54	
Pentachlorophenol	F	55	
Copper sulfate Pentahydrate	F	56	

TABLE 10-2 (cont'd)

Chemical	Type of pesticide[a]	NUPAS ranking	Top 10 homeowner chemical?
Phorate	I	57	
Boric acid	I	59	
Picloram	H	60	
Safrotin	I	61	
Ferric sulfate	H	62	
Baygon(propoxur)	I	63	
Tebuthiuron	H	64	
Chlorothalonil	F	65	
Benomyl	F	66	
Maneb	F	76	Yes
Captan	F	88	Yes

[a]I-insecticide, F-fungicide; H-herbicide, R-rodenticide.

[b]Many high volume chemicals in the NUPAS listing contain 2,4-D. We combined them because EPA will combine their reregistration reviews.

TABLE 10-3

Insecticide Chemicals Used in Selected Locations in Boston, Massachusetts

Facility/Location	Chemicals
State facilities - four office buildings and one mental health facility	Acephate, Boric Acid, Chlorpyrifos, Diazinon, Malathion, Methoxychlor, Piperonyl Butoxide, Pyrethrins, Resmethrin, Terpene
Transit system stations	Chlorpyrifos, Pyrethrins
Public housing projects	Acephate, Baygon, Bendiocarb, Boric Acid, Chlorpyrifos, Diazinon, Methoprene, Propetamphos
Various park areas cared for by the Boston Parks and Recreation Department	Carbaryl, Metasystox, Methoxychlor
Federal office building	Amidinohydrazone, Boric Acid, Chlorpyrifos, Diazinon
Private multi-family building used for public housing	Chlorpyrifos, Pyrethrins
Restaurant	Chlorpyrifos
Discount department store	Chlorpyrifos
Hotel	Chlorpyrifos
Industrial workplace	Chlorpyrifos
Retail food store	Chlorpyrifos
Private office building	Chlorpyrifos, Pyrethrins
Airplanes	Bendiocarb
Sports arena (non-food areas)	Diazinon
Hospital	Amidinohydrazone, Bendiocarb, Boric Acid, Chlorpyrifos, Pyrethrins

TABLE 10-4

Herbicide Chemicals Used in Selected Locations in and Around Boston, Massachusetts

Facility/Location	Chemicals
State facilities - four office buildings and one mental health facility	2,4-D; DCPA; Dicamba; MCPP
State highway rights-of-way	2,4-D; 2,4-DP; Aminotriazole; Dalapon; Diuron; Fenac; Fosamine Ammonium; Tebuthiuron
Three utilities' rights-of-way	2,4-D; Ammonium Sulfamate; Dicamba; Fosamine Ammonium; Glyphosate; Picloram; Triclopyr
Railroad rights-of-way	2,4-D; Ametryn; Atrazine; Dicamba; Diquat; Diuron; Glyphosate; Triclopyr
Unspecified rights-of-way	Metolachlor
Various parks areas cared for by the Boston Parks and Recreation Department	Glyphosate

GAO/RCED-86-97 Nonagricultural Pesticides

TABLE 10-5

Rodenticide Chemicals Used in Selected Locations in Boston, Massachusetts

Facility/Location	Chemicals
State facilities - four office buildings and one mental health facility	Brodifacoum, Bromadiolone
Transit system stations	Brodifacoum
Public housing projects	Brodifacoum, Bromadiolone, Rozol
Various park areas cared for by the Boston Parks and Recreation Department	Brodifacoum, Bromadiolone, Diphacinone
Boston neighborhood rodent control program - 108-block residential area	Brodifacoum, Bromadiolone, Diphacinone
Federal office building	Rozol, Diphacinone
Private multi-unit building used for public housing	Brodifacoum
Restaurant	Brodifacoum
Discount department store	Brodifacoum
Hotel	Brodifacoum
Retail food store	Brodifacoum
Private office building	Brodifacoum

GAO/RCED-86-97 Nonagricultural Pesticides

Pesticides are evaluated by EPA on a case by case basis. A specific label must be approved for each pesticide and cannot be altered by state law or regulations. At a minimum, pesticide labels must include a description of the pesticide's composition, allowable uses, directions for use, warnings, and disposal procedures. If approved, the pesticide receives a registration number. The Administrator of EPA has final decision authority relative to pesticide registration applications.

Specific risk data required with each registration application include the pesticide chemistry, pesticide residue chemistry, human toxicology, reentry protection, spray drift, wildlife and aquatic organism toxicology, plant protection, non-target insect effects, and pesticide performance. Using this information, EPA makes a decision whether to certify based on a risk-benefit analysis. EPA may require additional information regarding the pesticide whenever necessary, even after the pesticide has been registered. Registrants who do not respond in a timely fashion risk the pesticide's removal from registration. As noted earlier, EPA is in the process of re-registering pesticides registered prior to the establishment of these standards.

EPA recently announced a new policy relative to implementation of pesticide risk-benefit analyses. In the past, EPA complied with the so-called "Delaney clause" of the 1958 Federal Food, Drug and Cosmetic Act which prohibits the presence of residues on processed foods of any pesticide found to cause cancer. Now, EPA has initiated a policy of permitting the use of carcinogenic pesticides if in performing the risk-benefit analysis, the associated risk is found to be negligible. (247)

EPA has banned the sale of four pesticides: chlordane, aldrin, dieldrin and heptachlor. Most uses of lindane have been cancelled, including its use in indoor fumigation devices. EPA also has banned the indoor use of two wood preservatives, pentachlorophenol and creosote.

PESTICIDE CONTROL IN MASSACHUSETTS

Approximately 8,000 pesticide products are registered in Massachusetts pursuant to the Massachusetts Pesticide Control Act (M.G.L. c. 132B) and regulations promulgated thereunder (333 CMR 1.00-10.00). The Massachusetts Department of Food and Agriculture ("DFA") implements the pesticide registration process. In addition the DFA enforces federal and state pesticide laws and certifies and trains pesticide applicators. The DFA has two units involved with pesticide use in the state:

(1) The Pesticide Board which serves in an advisory capacity, setting policies and developing and adopting pesticide regulations. A Special Subcommittee of the Pesticide Board registers pesticide products.

(2) The Pesticide Bureau which carries out the work of testing, record-keeping and field investigation of pesticide use.

The Division of Food and Drugs within the Massachusetts Department of Public Health provides pesticide risk assessments to assist DFA as part of the pesticide registration process.

Relative to enforcement, DFA's general mandate calls for the prevention of pesticide use which may cause unreasonable adverse effects. Specific enforcement actions include the inspection of pesticide producer establishments, inspection of points of sale with 130 or more dealers of restricted use pesticides, inspection of potential instances of pesticide misuse or use without necessary approvals, and consumer complaints.

In addition to after the fact enforcement, the DFA has begun to deal with pesticides proactively. For example, the DFA issued advisory statements on controlling cockroaches in nursing homes and has distributed educational pamphlets to the general public. Recently, the DFA issued another advisory statement relative to the use of pesticides in occupied rooms. This advisory statement relates to most indoor spaces but specifically excludes residential applications of pesticides. The statement focuses on the importance of evaluating areas during pesticide application and allowing an adequate reentry period before the space is again occupied.

Pesticide use in restaurants and other food establishments is regulated specifically under 105 CMR 590.00, part of the Minimum Sanitation Standards for Food Establishments. Pesticide treatment must not result in contamination of food, food contact surfaces, or potable water supplies.

LOCAL CONTROL OF PESTICIDES

Under M.G.L. c. 111, Sec. 31C, boards of health have the authority to regulate and control atmospheric pollution including the emission of particulate matter, toxic substances, fumes and vapors, as may arise within the board's jurisdictional bounds. This authority may extend to the control of pesticides although any local regulation cannot conflict with state law. In the Town of Wendell v. Attorney General, a local board of health regulation was struck down for this reason although the court did not find that state pesticide law precludes local boards of health from formulating pesticide regulations which do not conflict with state law. (248)

Chapter 11: AIR IONIZATION

Hot dry winds such as the Sharav winds in Israel, the mistral winds in Southern France and the Santa Ana in southern California are known to have physical and psychological effects on humans. Those exposed to these winds suffer headaches, nervousness, depression and other ailments. (84)

Such winds are believed to produce changes in the electrical balance in the air, leaving some gas molecules positively charged and others negatively charged. Other causes of air ionization in addition to high winds are lightening, cosmic rays, sunlight, and radiation from radioactive materials.

Ions are unstable and thus short-lived in the atmosphere, however carefully controlled experiments have shown biological effects of these ions. One such study showed that serotonin levels in the blood and brains of mice increased with exposure to positive ions and decreased due to negative ions. (249) Increased serotonin levels cause sleeplessness, irritability, stomach upset and breathing difficulties. Current theory holds that serotonin is the root of air ionization's ill effects in humans.

To combat the effects of positive ions, negative ion generators have been used. Several reports of successful negative ion therapy have been produced. In one case negative ion therapy relieved migraine headache sufferers and in another cured infant asthmatics. Burn patients healed more quickly with less scarring, apparently due to negative ion exposure. (84)

It has been suggested that the quantity and quality of air an individual breathes influences changes in arterial walls and that the total quantity of negatively charged ions in the air will help maintain a proper arterial charge. Loss of normal net negative charge in arterial walls has been implicated as a key factor in the development of artherosclerosis. (250) Much more study is needed to ascertain the actual effect(s) of negative ion generators on human health.

COMMISSION RECOMMENDATIONS AND CONCLUSIONS

The indoor environment once was assumed to be a haven from outdoor air pollution. Recent evidence of indoor pollutant levels exceeding outdoor levels raises serious concern about health threats in indoor environments. The fact that individuals spend far more time indoors than outdoors only heightens this concern.

The Massachusetts Legislature recognized the importance of addressing the indoor air pollution problem and in 1987 created the Special Legislative Commission on Indoor Air. The Commission's first goal entailed the identification of health risks posed by various indoor air pollutants, acting either individually or cumulatively, and methods to avoid or minimize these risks. Based upon this information, the Commission has made important recommendations about how state government should address these health risks.

Options for managing these risks range from educating the public about indoor air pollution and appropriate mitigation measures to setting specific emission limits for each type of pollutant. The Commission proposes recommendations that constitute an overall strategy for dealing with the indoor air pollution problem.

JURISDICTION OVER INDOOR AIR QUALITY

Before responsibility for regulating indoor air pollution may be apportioned among state and local authorities, a firm definition of indoor air pollution must be established. The Commission has its own working definition for indoor air pollution: that condition in which indoor airborne contaminant levels either individually or in combination are known to or may adversely affect the health, safety and well-being of human occupants. Such airborne contaminants include radon, formaldehyde and other volatile organic compounds, asbestos, environmental tobacco smoke, biologicals, pesticides, combustion products, leaded paint dust and air ionization.

Numerous state agencies currently share responsibility for indoor air quality. The Commission recommends that specific areas of authority and responsibility be defined to ensure a comprehensive approach with minimal overlap of effort. To this end, an Interagency Coordinating Council should be convened, consisting of the Commissioners from DEQE, DPH, DLI, the Building Commission, the Office of Consumer Affairs and the Executive Office of Energy Resources. This council would address issues of interagency responsibility for public health and consumer protection from indoor air pollution and define appropriate state actions for prevention and remediation of indoor air health threats.

PUBLIC EDUCATION

Awareness of specific indoor air pollution problems has only recently emerged among the general public, with most attention concentrated on asbestos, radon and environmental tobacco smoke. The public is largely unaware of the general health threats posed by pollutants emitting from building materials, furnishings and other chemical and biological substances commonly found indoors.

The Commission recommends a strong public education initiative on the subject of indoor air pollution and indoor air contaminants, their sources and their effects on human health. This education effort must then go one step further by describing how pollution of indoor air by specific pollutants may be avoided entirely or may be mitigated when avoidance is not possible. The Commission is especially concerned that the public is educated about how to achieve and maintain adequate ventilation in new, existing or renovated buildings. Wherever possible, the public should be notified about conditions which may lead to indoor air pollution, such as where smoking occurs or where high radon levels have been detected.

The Interagency Coordinating Council shall designate lead agencies responsible for preparing and distributing public information about each pollutant type found indoors. Educational booklets on the subject of indoor air pollution should be made available, especially to prospective homebuyers.

The Commission further recommends that the subject of indoor air pollution be incorporated into school curricula as well as training programs for local building inspectors and board of health officers.

The Commission's educational recommendations for specific pollutants are as follows:

Asbestos

The Interagency Coordinating Council's designated lead agency shall provide reliable information and assistance to interested individuals about proper abatement techniques for asbestos. This agency shall compile a booklet in which all state laws, regulations and policies applicable to asbestos are summarized. Local boards of health should be notified prior to implementation of any asbestos abatement projects, in addition to those projects for which notice is already required under the State Sanitary Code. Information should be made available concerning alternatives to bonding for financing asbestos abatement projects.

Pesticides

Public notice should be provided when pesticides are being applied indoors so that occupants are not only aware of the pesticide use but may avoid application areas. Such notices should instruct the reader where more information may be obtained about the pesticide being applied. The

PROPOSED LEGISLATION AND REGULATIONS

The Commission recognizes the need to outline a statewide strategy for managing the indoor air pollution problem. This strategy should not necessarily emulate the approach taken for controlling outdoor air pollution since the indoor environment presents a unique regulatory challenge. The Commission has proposed specific legislation to implement this strategy.

A legislative resolve, creating the Interagency Coordinating Council on Indoor Air Quality has been drafted and the Commission recommends its passage. This Council would address issues of interagency responsibility for public health and consumer protection from indoor air pollution. The council would designate lead agencies to educate the public about the health threats associated with various pollutants and how they can be mitigated. The primary purpose of the Council would be to define appropriate state actions for the prevention and remediation of indoor air health threats.

Legislation has been drafted to require inclusion of adequate ventilation systems in new or renovated buildings and to establish a thorough ventilation system maintenance program. The Commission acknowledges the need for better ventilation and the need for energy efficiency. Balance between these two goals must be attained; ventilation systems using energy recovery technology can supply that balance.

Legislation has also been proposed relative to radon, requiring disclosure of any radon testing that has been done in a building at its point of sale. This legislation provides an incentive for the sellers to test by protecting them from future liability from the health effects of radon. No proposal for requiring radon testing has been set forth given the technical difficulties involved in such testing and the lack of a suitable contingent of qualified testers. The Commission does not believe mandatory tests would provide reliable information to the public; however, radon testing should be done wherever possible.

Several pieces of legislation have been filed relative to ETS. The Commission fully supports ETS legislation which regulates smoking in retail outlets and workplaces, restricts smoking in hospitals and limits access to cigarettes by minors. Pending pesticide initiatives facilitate the use of integrated pest management (IPM) and the reduced use of pesticides in indoor environments. The Commission supports reduced indoor use of pesticides.

The Commission recommends additional legislative measures to provide the means for state monitoring of the asbestos abatement industry. State financial assistance for schools faced with asbestos contamination must continue. Existing state regulatory programs have been successful in addressing asbestos contamination problems but need more staff and funding given the magnitude of the problem.

The Commission seeks to establish requirements for passing information about building operations and design parameters from the builders to the owners and operators as well as to subsequent owners and operators. Where a building or some portion of a building undergoes a change in use, the Commission recommends stricter enforcement of the Building Code which requires that the original ventilation system be reviewed to assess its adequacy and modified to meet the ventilation needs of the new use.

Commission recommendations for regulatory revisions focus on two areas: specific pollutant types and revisions to the state Building Code. For pesticides, the Commission recommends the Department of Food and Agriculture's "Advisory Statement Relative to the Use of Pesticides in Occupied Rooms" be incorporated into state regulations. Stronger enforcement mechanisms for controlling pesticide use is also warranted.

The Commission calls for regulations addressing microbiological pollution. The Commission believes proposed legislation mandating stricter ventilation standards represents the best available means for controlling indoor biological pollution.

The Commission believes VOCs should be controlled at the source; that is, within consumer products, rather than establishing specific exposure levels. At a minimum, use of VOC-emitting products must be discouraged. All consumer products should be designed to reduce VOC emissions and research should be conducted to identify alternate products that do not emit VOCs.

The Commission recommends the formulation of specific criteria for the design, fabrication and utilization of new or renovated buildings and furnishings to ensure safe indoor air quality. Proposed revisions to the State Building Code will be the first step toward meeting this goal. Specific legislative proposals were discussed above, involving major changes to the Building Code's ventilation system requirements for all buildings.

FUNDING PROPOSALS

The Commission made every effort to create and recommend options for controlling indoor air pollution which did not entail the expenditure of public funds. As a result, in order to implement the Commission's recommendations, new funding is needed only for the following proposals:

(1) Establishment of training programs for local boards of health relative to indoor air pollution issues including preventative and abatement measures. Additional resources are needed for laboratory support for local boards of health;

(2) Establishment of comprehensive programs for local building inspectors relative to the design, operation and maintenance of ventilation systems, as these systems relate to indoor air quality problems;

public also needs more instruction about the dangers involved with the misuse of over-the-counter pesticides.

Formaldehyde

The Interagency Coordinating Council's designated lead agency shall prepare and distribute public information that provides a general description of formaldehyde, its health effects and proper abatement techniques for formaldehyde contamination.

Environmental Tobacco Smoke

DPH efforts to educate the public about the dangers of smoking must be continued and strengthened in order to discourage nonsmokers from developing the smoking habit. Public notice should be posted in all public areas where smoking occurs. The Commission joins the DPH in its recommendation for the year 2000: All individuals through the course of their normal daily activities should be able to breathe ETS-free air.

Radon

The Interagency Coordinating Council's designated lead agency shall educate the public about radon and options for mitigating elevated radon levels in buildings. The uncertainty and limitations involved with radon mitigation must be fully disclosed. The lead agency must also issue a public advisory warning that radon is not just a problem in single-family homes but may occur in other private or public buildings.

TECHNICAL STANDARDS OF ACTION

Based on current understanding, standards for action to prevent, mitigate and eliminate indoor air pollution problems must be developed. Some exist already such as those for asbestos- and formaldehyde-containing products. The Commission's proposals include general as well as pollutant-specific standards of action.

Current statewide technical standards need to be revised based upon what has been learned about indoor air pollution and its prevention. The State Building Code must be revised so that provision of adequate ventilation is given the same level of consideration traditionally given to plumbing and utilities installation and operation. For the general control of all indoor air pollutants, the Commission supports the incorporation of Table 2 from ASHRAE 62-1981R into the State Building Code. This table prescribes outdoor air requirements for ventilation in commercial buildings. For residential buildings, the Commission recommends requirement of a continuous outdoor air ventilation rate of at least 0.5 air changes per hour. Provisions for requiring adequate ventilation system maintenance over the long term must also be incorporated into the Building Code.

The Commission supports changes in some pollutant-specific standards. For formaldehyde, the Commission recommends that the DPH review the current formaldehyde action level of 0.1 ppm and recommends appropriate legislative change if deemed necessary. In order to facilitate the development of technical standards for pesticides, the Commission supports efforts to estimate cumulative pesticide exposure levels found in indoor environments.

No recommendations are set forth for radon technical standards, although the Commission believes Massachusetts' participation in EPA's radon survey program should continue to gather baseline data. This information will be helpful in determining what standards ultimately may be appropriate for radon. Radon testing should be conducted in hospitals, nursing homes and schools. Individuals engaged in radon testing and mitigation must be certified. The Commission recommends that the State Building Code be revised to include building design standards for avoiding radon intrusion into new or renovated buildings.

The Commission recommends further research relative to biological contamination to identify, at a minimum, better monitoring techniques and to establish testing protocols. A comprehensive data base must be compiled of known biological pollutants and their health effects.

No emission limits have been set for individual indoor air pollutants. Any such effort would be fraught with uncertainty given the complexities of the indoor environment. Diverse pollutant sources exist, complicated by varying pollutant emission, reaction and ventilation rates. For these reasons, the Commission has chosen to focus on source control and proper ventilation standards rather than attempt to establish such emission limits.

Air quality standards and certification programs for individuals engaged in testing and mitigation for indoor air pollution problems should be established wherever possible and needed. Ensuring access to qualified remediation contractors and techniques is a major concern of the Commission as well as implementation of measures to improve the availability of state-certified testing programs and protocols. Consultants and abatement contractors for both public and private buildings should also be certified. The Commission does not support any actions that will inadvertently reduce the number of qualified workers available to handle these problems by creating overwhelming expectations of the consultants or contractors or by causing liability insurance difficulties.

The Commission has proposed legislation and regulatory changes requiring all new or renovated buildings to meet improved state ventilation standards. The responsibility for maintaining ventilation systems should pass from the installer to the building owner and subsequent owners. With the exception of residential buildings containing three or less units, ventilation records for such systems should be maintained and made available to current tenants and inspectors. Where there have been investigated incidences of indoor air quality problems, related records and reports shall be maintained for a reasonable period of time.

(3) Development of a DPH program to track statewide biological contamination research efforts. The DPH also needs funding to continue gathering data on the health effects associated with biological contaminants, conditions under which such health effects may occur and methods for controlling contamination;

(4) Provision of a State Building Commission staff person with expertise in the areas of heating, ventilation, and air conditioning systems and how these systems relate to public health issues; and

(5) Increase in the number of state level staff in asbestos programs. Additional state funding must be provided so that the Commonwealth will meet the requirements of the federal Asbestos Hazard Emergency Response Act.

CONCLUSION

Indoor air pollution seriously threatens public health. Scientific testimony and information provided to the Commission shows that many diseases and symptoms are attributable to indoor air pollution: irritation of the eyes, nose and throat, headaches, fatigue, nausea, asthma, emphysema, pneumonia, lung cancer, heart disease, chemical sensitivity, liver and central nervous system damage and many other ailments. As a result, billions of dollars are spent annually on pollution abatement and health care costs resulting from indoor air pollution. In response to these problems, the Commission's recommendations embody broad policy and technical guidance designed to combat indoor air pollution. Proposed legislation and strengthened state regulations will protect individuals from serious and avoidable indoor air health threats and avoid significant health costs. The Commission's proposed public education initiative will equip individuals with the knowledge needed to minimize indoor air pollution through careful choice of building materials and consumer products. As a greater understanding of the indoor air pollution problem is achieved, the state and its new Interagency Coordinating Council must be ready to review these recommendations and consider new approaches for controlling the health threats of indoor air pollution.

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